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VOLUME 12 NUMBER 6

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DECEMBER, 1924

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO, ILLINOIS. ANNUAL SUBSCRIPTION, $8.00

Entered as Second-Class Matter, Jan. 7, 1919, at the postoffice at Chicago, Illinois, under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of Oct. 3, 1917, authorized Jan. 15, 1919.

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Archives of Neurology and Psychiatry

Vol. 12 DECEMBER, 1924 No. 6

LESION OF LEFT LENTICULAR NUCLEUS AND ADJACENT STRUCTURES

APHASIA, BOTH "MOTOR" AND "SENSORY." SIGNIFICANCE OF THE FINDINGS FOR THE INTERPRETATION OF APHASIA *

FRANCIS X. DERCUM, M.D.

PHILADELPHIA

It will be recalled that shortly before the war aphasia was the subject of active discussion. This discussion had been precipitated in 1906 by the publication of the views of Marie. These views were novel and in a sense iconoclastic. They aroused decided opposition, notably on the part of Déjerine, and for a time a veritable conflict existed between two groups of neurologists who arrayed themselves on opposing sides. Numerous papers on aphasia appeared both in France and elsewhere. It was discussed at the meeting of the International Medical Congress at Buda-Pest in 1909, at the meeting of the International Medical Congress at London in 1913, and in February, 1914, the Philadelphia Neurological Society devoted an entire evening to the subject. Subsequently interest in aphasia was less active until the appearance of the important papers by Henry Head in Brain in 1920 and 1923.[1]

My own interest in the subject has again been aroused by an observation made in my hospital service during the past year, which again forcibly directed my attention to the views of Marie. The case is briefly as follows:

REPORT OF A CASE

History.—J. MacM., a white man, aged 52, a painter, was admitted to the ward of nervous diseases of the Jefferson Hospital, Feb. 28, 1923, evidently suffering from a stroke of paralysis; there was loss of power in the right side of the body, and he was unable to talk. The following history was obtained from a brother.

The father died of tuberculosis; the mother was dead, cause and age not known. One brother died of apoplexy; a sister was said to have died of acute miliary tuberculosis; two brothers were living and well.

The early childhood history was unknown. The patient had had typhoid fever at the age of 15, followed by an ulcer on the cheek, which persisted about

* Read at the Fiftieth Annual Meeting of the American Neurological Association, Philadelphia, June, 1924.

1. I unfortunately have not had access to the publications of Henschen.

a year. Two years before examination he had heart trouble, which kept him in bed for five weeks; he had difficulty of breathing, and his feet and ankles were swollen. He had been a moderate user of alcohol. The brother did not know whether the patient had had any venereal disease. He had never had painter's colic. He had never had any operations or any injuries and had never had any difficulty with speech or walking. The patient had been seen by his brother a few minutes prior to the onset of the present illness. At that time, the patient was in his usual condition except that just prior to the attack, he complained of a slight headache and a severe cold. He was entirely rational, had no difficulty of speech and no apparent weakness on either side of the body.

The patient had been picked up unconscious on the street by the police. On admission to the hospital, he was unconscious and had a flaccid palsy of the right side. Some hours subsequently, he gradually regained consciousness with some return of power in the arm and leg. He was, however, unable to speak except to say an occasional "yes" or "no," and these replies were apparently not relevant to the questions asked. The right side revealed as at first noted a flaccid palsy with reduced reflexes. The pupils were equal, 4 mm.; reaction was normal. The optic disk was round, of good color, with a shallow cup, margins well defined and no evidence of pressure. There were no muscle palsies. The blood Wassermann reaction was negative. The spinal fluid, examined on March 2, revealed an increased pressure. It was clear, with a lymphocyte count of 1; the sugar was reduced, the globulin normal. There was no blood in the fluid and no organisms. The Wassermann reaction was negative. The urine revealed a specific gravity of 1.031; no albumin, no sugar, no formed elements.

There was no noticeable change in the condition of the patient for a period of a week, when, on March 8, it was noted that the reflexes had become exaggerated, and that there was some increase of power in the arm and leg. The knee jerk was exaggerated, but there were no ankle clonus and no Babinski sign.

The condition remained unchanged until March 14, when the patient was noticeably weaker. Loss of speech was complete. He died on March 16 from bronchopneumonia.

The interest in the foregoing case centers of course in the aphasia. At no time that the patient was under my observation could he comprehend anything that was said to him, and the result was the same when written or printed matter was held before his eyes. He evidently attempted to reply when forcibly spoken to, but the only answers received, as already stated, were "yes" or "no," and even these words were uttered imperfectly and were clearly not responsive to the questions asked.

Necropsy revealed a hemorrhage in the left hemisphere which had involved extensively the lenticular nucleus, the internal capsule and the white substance subjacent to the convolutions of the island of Reil. The lesions extended sufficiently far posteriorly to invade the anterior portion of the temporoparietal isthmus. It is to be noted, also, that, as revealed by the change in contour of the brain, there was considerable loss of substance. The third frontal convolution, together with its subjacent white matter, was entirely free from involvement. (See illustration).

STRUCTURES INVOLVED IN APHASIA

How are we to interpret the findings in this case? Marie, it will be recalled, rejected the division of aphasia into motor and sensory, and substituted therefor the conception of aphasia as a clinical whole, a unity, the basic symptom of which he regarded as an impairment or loss of the power of speech comprehension. To this basic symptom and common to all cases, there was added in varying degree an impairment of emissive speech. To him, Broca's aphasia so-called consisted of these two factors. The failure of the power of language comprehension, he ascribed to lesions of the zone of Wernicke. The failure of emissive speech, he ascribed to lesions of the lenticular zone. Pierre Marie gave the name lenticular zone to a quadrilateral which was defined anteriorly by a vertical plane passing transversely through the anterior marginal fissure of the insula and posteriorly by a parallel plane passing through the posterior marginal fissure of the insula; within it was bounded by the lateral ventricle and without by the surface of the insula. Above it extended to the superjacent convolutions, below to the subthalamic region. According to this conception, the third frontal convolution and its subjacent white matter is excluded. Posteriorly the quadrilateral is in relation with the zone of Wernicke through the temperoparietal isthmus. Full recognition was of course accorded to the fact that this zone contains many different structures, namely, the cortex of the insula, the white matter subjacent to the latter, the claustrum, the external capsule, the lenticula, the internal capsule, the caudatum, a large portion of the thalamus, the superior longitudinal fasciculus and other structures as well. It is quite evident that the thalamus is not included in the vascular lesions ordinarily occurring. The lesions involve, as is well known, the internal capsule and to a varying degree the lenticular nucleus, and it may be, as in the present case, also the external capsule and the insula. That the corona radiata and the radiations of the corpus callosum may be involved in accordance with the extent of and severity of the hemorrhage, goes without saying. Structures possessing very different functions may therefore be involved. Many of them, however, may be dismissed from our consideration without hesitation.

If we are in search of an explanation for the cause of the motor phenomena in a given case, we are naturally referred to structures having definite relations to motor functions. Two such structures present themselves; first, the cortical motor apparatus with its projection fibers, and second, the striatum with its emissive pathway, the pallido-rubrospinal tract. Marie focused his attention on the striatum, more particularly on the lenticula. Here analogies between the anarthrias of pseudobulbar palsy and the anarthrias encountered in so-called motor aphasias at once suggest themselves. Marie was strengthened in his view

in regard to the importance of lesions in the quadrilateral by the not infrequent finding of such lesions in so-called motor aphasia, the third frontal convolution being at the same time intact. Such findings were reported by himself, by Souques, by Moutier, by myself and by others.

The Striatum.—Our increasing knowledge of the striatum justifies, I believe, definite conclusions as to its functions, and among these to the rôle which it may play in speech disturbances. In order to make my meaning clear, a brief digression is necessary. The brain stem—namely, the medulla, pons, crura, thalamus and striatum—is frequently and properly spoken of as a segmental apparatus, just as we apply the conception of a segmental apparatus to the spinal cord. It has definite segmental relationships. The end of the primitive neural tube, the cortex of the neo-encephalon, has no segmental relationships. Its neurons in their development can only establish relationships with or through the primitive segmental brain, the paleo-encephalon; ingress and egress are possible only through the latter. Not having segmental relationships, the neurons of the neo-encephalon are necessarily limited to the function of intercalary neurons. If the neo-encephalon has grown in response to the stimulus of function—and the facts of embryology, comparative anatomy and paleontology show that it has so grown—it means that a multiplication of intercalary neurons has taken place and, as a corollary, an increasing adaptability of response to the impacts received from the outside world; that is, there is present in the neo-encephalon a wide range of variability in the responses; in other words, the associations of the ten thousand million neurons of the cortex are variable, not fixed. New factors in the environment, new impacts transmitted through the paleo-encephalon, the brain stem, result in new associations, new adaptations of responses. Fixation is absent.

When we turn our attention to the brain stem and spinal cord, an entirely different situation confronts us. In the spinal cord, for instance, the purely automatic character of the responses must be admitted without question. The responses are definite and invariable. The synaptic associations between the neurons concerned have in the course of time become fixed. A similar interpretation must necessarily be extended to the medulla, the pons, the crura and the striatum. Here fixation and invariability of response likewise obtain. It is quite possible that early in the evolution of vertebrate forms the neurons of the striatum possessed a limited degree of variability of adaptation. Possibly it still presents such limited variability in some reptiles and in birds, but in the higher animals that variability of adaptation has become a function of the great usurper, the cerebral cortex. What then is the function of the striatum? Evidently it is concerned with

fixed responses, i. e., with fixed neuron associations.[2] It would appear that primitive motor functions, such as standing or walking, which depend on constantly recurring neuron associations, are early relegated to the striatum. Standing and walking are early performed without conscious effort. The same is true of especially acquired functions, such as swimming. The swimmer first establishes the associations concerned in the act of swimming by movements consciously performed, by movements the correct performance of which depend on consciousness and attention. Soon these complex movements are performed automatically and subconsciously. The complex associations required in the necessary daily acts of life, such as dressing and undressing, in the use of utensils or in the playing of musical instruments, are examples open in greater or lesser degree to the same interpretation. Surely this interpretation applies with especial force to the highly complex associations concerned in speech. At first, during the period in which speech is being acquired, the effort is conscious and cortical. Those of us who have acquired foreign languages after the period of childhood can, I think, give convincing testimony as to the conscious effort required. Soon, however, all sense of effort ceases, and words, phrases and sentences are uttered automatically and spontaneously. The cortex early ceases to concern itself with the associations required, and, like standing, walking, swimming and the movements concerned in the playing of instruments, these associations are relegated to the striatum. The neurons of the cortex have primarily, as we have seen, the function of variability, of adaptability of response. Responses recurring with relative frequency soon become a fixed function, and are then automatically assumed by the lower and more primitive mechanism, the striatum.

The disturbance and loss of emissive speech due to a lesion of the lenticula in the present instance becomes therefore comprehensible. Probably, too, it is the putamen which is more directly concerned than the pallidum. This would be in harmony with the later development of the putamen.

Perhaps the interpretation here advanced becomes clearer when we pause to reflect that all of the voluntary muscles are subject to a double innervation; first, an innervation derived from the primitive motor apparatus of the paleo-encephalon, the brain stem, and which we now speak of collectively as the extrapyramidal innervation, and second, the innervation derived from the motor area of the cerebral cortex. The reciprocal interplay of these two sources of innervation is grossly illustrated in the phenomena presented when one of these is impaired or destroyed. For example, in hemiplegia due to a lesion of the internal

2. Dercum; Physiology of Mind, Philadelphia, W. B. Saunders Company, 1922, pp. 55 and 56.

capsule, the resulting spasticity of the paralyzed side is due to the now dominant innervation of the muscles by the motor apparatus of the paleo-encephalon—the striatum and its adjuncts. On the other hand, when the motor apparatus of the paleo-encephalon is involved, as we find it in paralysis agitans and the parkinsonian symptom-group generally, the muscles are given over to the now dominant innervation of the motor area of the cerebral cortex. The fact of this double innervation of the striated or voluntary muscles is to me one of great significance. It bespeaks a constant interplay of the two sources of innervation. The activity of the motor area of the cortex in pursuance of its special function for the formation of new and adjustable responses leads to new movements, new associated and combined actions of muscles. These new movements cannot take place, however, without the interplay, without the concomitant cooperation, of the innervations derived from the striatum. The striatum necessarily plays its share in the changes of the muscle tonus involved. We commonly, perhaps almost subconsciously, regard the striatum—in a large measure at least—as a vestigeal structure. The term paleo-encephalon while appropriate and abundantly justified morphologically, is a trifle misleading, for the striatum is not wholly an ancient structure. The facts show that like the cortex of the cerebrum it has grown in size as we ascend in the scale of vertebrate morphology, and it has evidently increased as the demand for associated movements, more and more complex, has increased. These movements are more numerous and more complex in the reptile than in the fish, more numerous and more complex in birds than in reptiles and most numerous and most complex in mammals. It can be fairly claimed that the striatum reaches its fullest development in man, in whom the number and complexity of associations reaches its highest expression. In fact, it is this gradual and increasing development of the striatum which has caused, as is well known, the term neostriatum to be applied to its later developed portions, namely, the putamen and the caudate nucleus, while the term paleostriatum has come to be applied to the earliest and most primitive portion, namely, the globus pallidus.

Cooperation Between Striatum and Motor Area of Cortex.—How is the cooperation between the two sources of motor innervation, the striatum and the motor area of the cortex, maintained? There is little or no direct connection between these two structures; or, at most, if such connection exists, it is admittedly slight. The thalamus, on the other hand, has exceedingly intimate connections both with the cortex and with the striatum. Doubtless impacts—sensory impressions—received from without, reaching the thalamus, are transmitted by the latter to the cortex. A pathway of transmission having been estab-

lished through the latter, the emissive area—the motor area—is finally reached.[3] An impulse now finds its outward pathway through the cortical motor projection fibers. But this is not all; the fibers which connect the thalamus with the cortex are not only thalamocortical but corticothalamic as well, and impulses are transmitted from the cortex also to the thalamus, and from the thalamus through the thalamostriatal fibers directly to the striatum. Assuredly we have here a mechanism which abundantly explains the cooperation between the two sources of motor innervation—the cortex and the striatum—a cooperation which, when we consider the facts of morphology, physiology and neuropathology, becomes an inescapable inference.[4]

CAUSES OF APHASIA

Let us now turn our attention to the underlying factor of aphasia. What this underlying factor is depends on our point of view. We have already learned that to Marie the basic factor of aphasia is the loss of the power of speech comprehension, and to this interpretation I shall presently return. Clearness requires that I first consider briefly the older and what may be termed the conventional view and of which Déjerine, among the most distinguished neurologists of our day, was for a long time the outstanding exponent. As illustrative of his position, Déjerine's[5] detailed description will serve our purpose best, as this description antedated the later appearing discussion with Marie by a period of five years. Further, the accuracy of Déjerine's descriptions admits of no question. We will find also as we progress that his difference of opinion with Marie was based not so much on difference of fact as on differences of point of view and of approach to the subject. As is well known, Déjerine separated aphasia into two forms, namely, "motor aphasia or aphasia of expression" and "sensory aphasia or aphasia of comprehension." He further divided motor aphasia into true or cortical motor aphasia and pure or subcortical motor aphasia. The latter, due entirely to a subcortical lesion, is featured by an inability to utter words, "of articulating sounds in all their modes, but all of the other modalities of speech are preserved."[6] In other words, the patient

3. Dercum; (Footnote 2) pp. 59 to 62.

4. It should perhaps be added that Ramon y Cajal regards the relations between the motor area of the cortex and the striatum as direct, these relations being established by collaterals from the pyramidal fibers. Herrick: Introduction to Neurology, Philadelphia, W. B. Saunders Company, Ed. 3, p. 186.

5. Déjerine: Sémiologie du système nerveux, in Bouchard, C.: Traité de pathologie générale, Paris, Masson et Cie **5**:1901. Tr. Internat. Med. Congress, London, 1913, Section on Neuropathology, p. 85.

6. Déjerine: (Footnote 5) p. 402.

in this form comprehends in a perfectly normal manner both spoken and written language, writes perfectly both spontaneously and at dictation, and unmistakably has a clear idea of the words he wishes to utter. The sole symptom consists in the inability to utter the words. I presented such a case before the Philadelphia Neurological Society in November, 1913.[7] Whether such a symptom should be regarded as a true aphasia or should be regarded as merely an anarthria depends on the meaning we place on the word aphasia. It is significant perhaps that Déjerine spoke of the other form of motor aphasia, Broca's aphasia, as *true* aphasia.

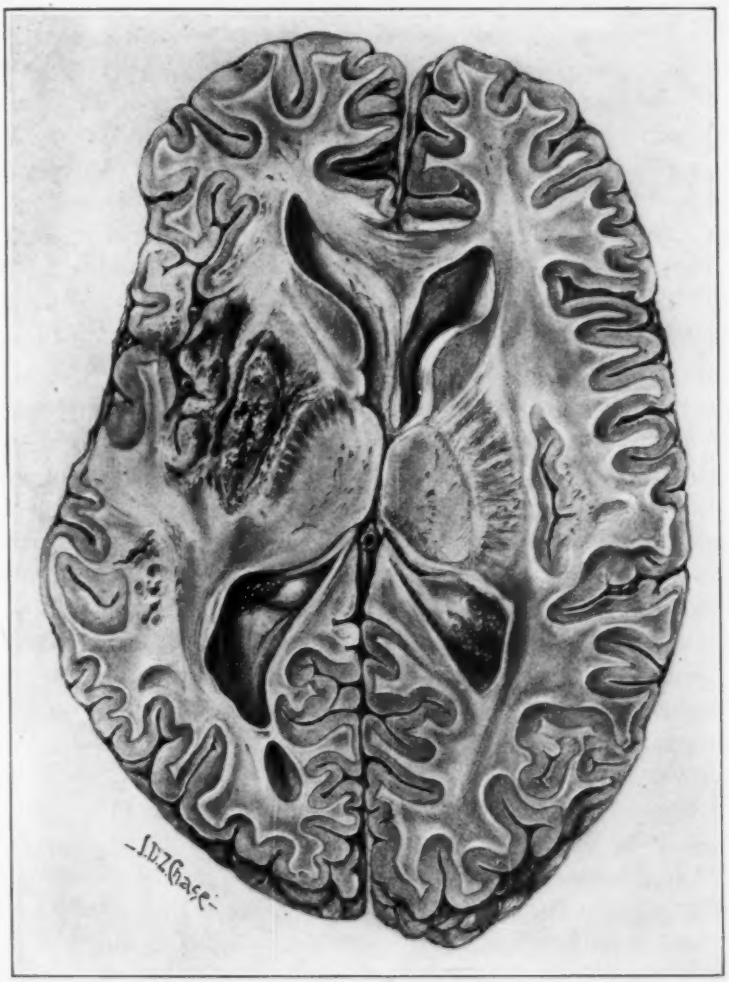
Motor Aphasia.—Let us now turn our attention to this other form. This, let us repeat, Déjerine spoke of as "true cortical motor aphasia or aphasia of Broca."[8] Its symptoms may be briefly summarized as follows: First, the patient "has lost the ability to translate his thoughts into speech." Second, this loss is not absolute; quite commonly there is retention of words like "yes" and "no," together with occasional other words or phrases, oaths, expletives, and the like. Third, the disturbances of speech are not limited to the loss or impairment of emissive speech, but disturbances of speech comprehension are also present. Déjerine pointed out that in this form of aphasia there is always present a disturbance of the ability to read. "Mental reading is always changed." He pointed out further that the comprehension of spoken language is also involved although this involvement is always less than that of reading. It is, however, an invariable accompaniment. He expressed himself as follows: "Although in cortical motor aphasia the disturbances are manifestly more accentuated on the side of spoken speech and of the written language, *all of the modalities of language are involved*. This fact he emphasized by expressing it in italics. "The varieties," he said, "in accordance with the degree of this alteration, are very numerous, but we always meet with this law; change of all the modes of language with predominance on the side of articulate speech."

Sensory Aphasia.—Let us now turn our attention to the form which Déjerine called sensory aphasia or aphasia of comprehension. Déjerine[9] here said:

> In motor aphasia, the disturbances observed predominate on the side of the articulation of words. The lesion involves the center of the motor images of articulation. Here, on the contrary, it is the centers of reception which are the seat of lesions, and in keeping with this the comprehension of speech and of reading is impaired. Are there one or more sensory aphasias? For the sensory

7. Dercum, F. X.: J. Nerv. & Ment. Dis., 1914, p. 137.
8. Déjerine: (Footnote 5) p. 399.
9. Déjerine: (Footnote 5) p. 404.

aphasia of Wernicke, Kussmaul has substituted two clinical forms; word blindness and word deafness, recognized also by Charcot. Wernicke never ceased to oppose this division. The most recent clinical and anatomicopathologic studies are in agreement in demonstrating the correctness of the conception of Wernicke.



Lesion of the left lenticular nucleus, internal capsule and white matter subjacent to Island of Reil.

There is only one sensory aphasia, comprising at one and the same time the disturbances of the comprehension of spoken speech and the disturbances of reading.

Word deafness is the symptom which is the most striking. The patient, whose auditory acuity is normal, is incapable of comprehending words spoken before him. He resembles an individual transported into a foreign country, the language of which he does not understand. The words strike his ear as different

and varied sounds, but not as representing ideas. The patient has then the appearance of a deaf person; the verbosity of his language, the incoherence of the words which he utters, lead also to his being regarded as attacked by confusion or with mental alienation. The degree of the verbal deafness is, moreover, very variable, according to the case.

The fact that Déjerine recognized only one form of sensory aphasia should be especially emphasized. When we analyze Déjerine's description of sensory aphasia we find portrayed conditions strikingly identical with those observed in the aphasia termed true motor or Broca's aphasia. Thus, we note that Déjerine's description of the alexia present agrees, even in detail, with that given by him of the alexia present in Broca's aphasia. The alexia may be pronounced in either form or it may be so slightly marked in either form as to necessitate, both in sensory aphasia and in Broca's aphasia, care in the study of the case lest the symptom be overlooked.[10] The fact remains that it is always present in both forms.

Comparison of the Two Forms of Aphasia.—When we turn our attention to the comprehension of spoken speech, we note that failure of comprehension is present in both forms, more marked in the so-called sensory form, but none the less present in both. A comparison between the sensory phenomena of Déjerine's so-called true motor aphasia or Broca's aphasia and the sensory phenomena of so-called sensory aphasia or Wernicke's aphasia, fails to reveal any difference in kind, and if it be maintained that there is a difference in degree, this disappears when —as Déjerine himself pointed out—we study individual cases. If this be the fact, the essential identity of the sensory phenomena in Broca's and Wernicke's aphasias should be frankly admitted. Clearly, Broca's aphasia consists of a loss of language knowledge to which the symptom of the loss of emissive speech has been added; that is, to the phenomena of a so-called sensory aphasia there are added the phenomena of Déjerine's pure or subcortical motor aphasia or Marie's anarthria. So-called sensory aphasia, or Wernicke's aphasia, as it should properly be termed, merely differs from Broca's aphasia, if it differs at all, in that in it the loss of language knowledge is more profound; there is no difference in kind.

In a much more recent publication, a paper on aphasia, anarthria and apraxia presented at the section on neuropathology at the International Congress in London in 1913, Déjerine[11] drew a distinction between the alexia and word deafness present in Broca's and Wernicke's aphasias. He said "In motor aphasia, aphasia of expression, the com-

10. Déjerine: (Footnote 5) pp. 400 and 406.

11. Déjerine: Part I, Section of Neuropathology, p. 85.

prehension is disturbed only for the images, which, in the genesis of language, are subordinated to the motor act, that is to say, the visual images."[12] Similarly, he said: "As to the disturbances of the comprehension of spoken words which motor aphasics present . . . they are not all those of verbal deafness. They consist of a defect of evocation of the verbal auditory image." He did not tell us what he meant by the visual image being "subordinated to the motor act," nor how nor why this subordination gives rise to alexia. Again, he did not explain in what respect the "defect of the evocation of the verbal auditory image" differs from the same defect in sensory aphasia. After all is said and done, the clinical fact of the loss in both forms remains.

Déjerine further pointed out in his sémiologie,[13] in speaking of so-called sensory aphasia, that the intelligence is always involved.

> By the simultaneous loss of the comprehension of spoken speech and of reading—verbal deafness and blindness, by the disturbances which they present on the side of spoken speech and writing—paraphasia and jargon aphasia, agraphia, these patients find themselves cut off from all communication with their fellows. The intellectual weakness in them is in general more marked than in the motor aphasic, but it may sometimes be almost nil. . . The intellectual deficit betrays itself in the mimicry, in the gestures, which are always less expressive than in the normal man; nevertheless, the sensory aphasic is ordinarily capable by means of gestures to make his desires and his thoughts understood, at least partially. There exist of course in this respect quite marked differences according to the case.

Finally Déjerine pointed out that in "total" aphasia, "the intellectual deficit is much more marked than in sensory or motor aphasia."

Marie has been much misunderstood abroad by his own use of the term intellectual deficit. Many years ago he pointed out that intellectual deficit manifests itself in three ways: First, in the deficit seen in cases of arrest and imbecility; secondly, in cases of mental disease in which dementia is present; and, thirdly, as seen in aphasia. The specific mental loss revealed by the aphasic may be complicated by a more general mental loss resulting from the wide extent of the lesion. As a matter of fact, both losses may in a measure be commingled. For instance, a man who was formerly skilful in performing the acts or duties of his special calling, may after an apoplectiform lesion resulting in aphasia be unable to perform these acts or duties as before. For example, a patient who during his normal period was a very skilful cook may make a gross and lamentable failure after he becomes aphasic, due, not to his loss of language knowledge, but to his general mental impairment.

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12. Déjerine: (Footnote 11) pp. 91 and 92.
13. Déjerine: (Footnote 5) p. 412.

MENTAL LOSS CAUSED BY APHASIA

It behooves us now to consider once more and briefly the nature of the specific mental loss revealed by the aphasia. This loss must, I think, be sought for in the loss of language comprehension. Language comprehension is in the normal person a fundamental function. Without it emissive speech cannot be developed. Of necessity, language comprehension antedates all other language functions. Of what does this language comprehension consist? Clearly the evolution of speech recognition or speech understanding must have been preceded by the evolution of a sense of simple sound recognition; that is, the first step must have consisted in the formation in the mind of primitive man or of his progenitors of an association of the sounds of nature with the objects or phenomena giving rise to these sounds, just as man has learned to associate the tactual qualities of the objects of nature with the objects themselves. The evolution of the faculty of sound recognition is, in a sense, therefore, analogous to the evolution of the faculty of stereognosis. In stereognosis there is an association of the tactual qualities, in sound recognition an association of acoustic qualities. In other words, to man's knowledge of the external world, made up of the perception of tactual qualities, there is added, among other things, a knowledge of the external world made up of its qualities of sound. The primitive recognition of natural sounds, that is, the association of the sounds of nature with the phenomena giving rise to them, is a faculty which man doubtless shares with other animals. It must of necessity, in addition, form the basic substratum on which in him the more complex function of language recognition has been built. Certainly it seems legitimate to assume that language recognition and language comprehension have been evolved from this faculty of the primitive recognition of sounds. In ordinary sound recognition, there is the immediate association of the sound with the corresponding object in nature. In the sound recognition of speech, there is the recognition of definite sounds associated not directly with objects in nature, but with the conceptions or memory pictures of these objects which the sounds call forth. Viewed in this light, the region of Wernicke becomes but a part of the great general posterior association area of Flechsig, and much of the mystery attached to the speech function disappears.

The steps by means of which spoken speech was evolved will probably never be known and will forever remain a matter of speculation. It is fair to assume, however—at least, it does no violence to any laws of probability—that the first attempts at spoken speech consisted of efforts on the part of man to reproduce sounds similar to those heard by him in nature. It would seem that in some such way as this notions

and ideas were in the beginning communicated to his fellows; no doubt vaguely and imperfectly at first. Based on such rudimentary beginnings, a progressive evolution of speech comprehension and spoken speech is readily conceivable. More and more would arise the tendency in man to reproduce the articulate sounds made by his fellows. Indeed, this is exactly what we see in the developing child. How complex the motor associations are that are required becomes apparent when we reflect that they include the innervations of the muscles of respiration, of the larynx, of the palate, of the tongue, of the buccal cavity and of the lips. Notwithstanding their complexity, these associations soon become as automatic as those concerned in walking. They become relegated to the field of those fixed or relatively fixed associations which it is the function of the striatum sooner or later to assume. It is to the failure of this function that Marie has applied the term anarthria.

When all is said and done, there can be no doubt as to the cortical nature of the function of speech. This must apply alike to the function of speech comprehension and to spoken speech. This qualification must, however, be made. Speech comprehension must of necessity antedate spoken speech. It is an unavoidable hypothesis that this has been the process in the evolution of the speech faculty in primeval man. As regards the acquisition of language by the child or of new languages by the adult, the facts admit of no reasonable doubt. As regards the emissive faculty of speech, it likewise must in its beginning have been cortical. The imitation of natural or articulate sounds must have been attended by a conscious effort. The necessary associations of neurons concerned in the multiple and complex coordinations required by emissive speech are accompanied in the acquisition of foreign languages by the adult, by an effort of will and persistence that leaves no doubt that the activities are cortical. Sooner or later it appears that both in the child and in the adult acquiring foreign languages, the associations become fixed and are relegated to the striatum, probably to the putamen.

In this way, the motor loss in the case here reported can be explained, while the loss of speech comprehension probably depends on the interference with the temporoparietal isthmus, i. e., with the inferior longitudinal fasciculus. Probably the rather marked loss of substance dependent on the severity of the lesion plays a rôle in the totality of the symptoms.

The case here reported because of the completeness of the loss does not justify a detailed discussion of cortical speech processes. However, a quotation from Head's paper [14] on speech and cerebral localization

14. Head: Brain **46**, Pt. 4, p. 355 (Nov.) 1923.

because of its bearing on the interpretation of the present case may be permitted. He says:

> The power to express ourselves in spoken words is acquired by voluntary effort, but ultimately becomes almost automatic. In the same way, when learning to play the piano, the fingers are at first laid laboriously on each key, and a complicated apparatus is thrown into action, which sounds the note. Soon, with practice, the fingers move automatically and consciousness is occupied in reading the musical symbols on the printed page. Finally, with increased facility, even this becomes almost automatic; the expert musician scarcely sees the page, for the nature of the musical phrase suggests to him the sequence of the accompanying harmonies.

DISCUSSION

Dr. Charles K. Mills, Philadelphia: I recognize fully the suggestive value of Marie's view reiterated and extended by Dr. Dercum.

A tendency is shown to use the term "conventional" with regard to certain theories concerning speech. "Conventional" in a case like this means a view or theory which is the result of years of study of a subject. I have never been shaken for a moment from my views—my extreme views as they are sometimes called—as to cerebral localization.

In connection with aphasia, without having time to go fully into this matter, I might say that I recognize and everybody who knows anything about brain function and has studied it clinically has recognized that a certain degree of mental impairment is present with a lesion of Wernicke's zone, or indeed a considerable lesion of any part of the brain.

It is putting the cart before the horse, however, in my estimation, to make mental impairment the first step in the explanation of this matter. The cases which really demonstrate the part played by Broca's convolution, to which I would add, and have added in my published views, the anterior part of the insula, have been numerous and convincing.

We hear much talk about association phenomena and association centers. Certainly there are developed association centers, but what does "association" mean? It means that something has been developed in the brain by association. Secondary centers have been formed as the result of association of primary centers.

My own views are those of Déjerine, with maybe some modification. A case like this (with all due respect to Dr. Dercum) seems to me to prove nothing with regard to the theories of aphasia. The man died in a short time; he tells us that the lesion involved was in largely different parts of the so-called "language area" of the brain. In every great lesion, and to some extent in every lesion of any part of the language area, all the factors of speech are involved.

I do not think that Marie or any of his followers has ever overturned the so-called "conventional" scheme of cerebral speech.

Dr. Walter F. Schaller, San Francisco: A noteworthy contribution to the subject of aphasia has been made recently by Henschen of Stockholm. This investigator has made a clinico-anatomic analysis of all available aphasia cases reported in the literature, numbering over 1,300. There are over 100 cases of insular and lenticular lesions alone. Henschen concludes that there are definite cortical speech centers which by their association form concepts, and finally ideation.

He believes that word deafness and word blindness are due to impairment of special brain centers which are separate and independent. He therefore opposes Wernicke. Although a bitter antagonist of Marie, because of his attempted revision of aphasia, he favors the idea that the lenticular nucleus has to do with speech defects, and in this view disagrees with Déjerine.

The anterior part of the putamen, according to the analysis, has to do with aphasia, whereas the posterior part has to do with dysarthria.

Dr. Michael Osnato, New York: The difficulty with the whole question of aphasia has been that attempts have been made primarily to find some place or places in the brain in a more or less schematic way, which would serve as a convenient explanation for the function of speech. Then there has been the second difficulty caused by lack of sharply separating anarthria or dysarthria from aphasia as disturbances of speech function.

In considering the question of aphasia, one must remember that, just as Dr. Dercum has said, speech has developed intimately with intelligence, and it seems to me just as futile to attempt to localize intelligence in any one place or in several places in the brain, as it is to localize speech sharply.

Even a cursory reading of Head's first contribution leaves one deeply impressed by the remarkably exhaustive manner in which he examined his cases clinically. His clinical studies led him to certain definite conclusions which in his second contribution he materially modified. He was definite in his first contribution, sometimes accentuated in his definiteness that there was no possibility of localizing speech; that speech could not be localized in circumscribed areas of the cortex. Then he proceeded to give us his more or less arbitrary divisions of the varieties of aphasia into the nominal, the semantic, the verbal, syntactic, etc. In his more recent publication, Head departed from that attitude and outdid all the localizers. He proceeded to change the localization of the various speech functions, and when his work is analyzed one is disappointed. His conclusions depend altogether on six cases. Necropsy was not performed in any of the cases. They were cases of craniocerebral injuries, which he localized briefly in this fashion. He had plaster models of the skulls of the patients who had craniocerebral defects, and accurately located the places on the skull corresponding with the areas of cortex beneath which he thought were injured, and on the basis of that rather doubtful sort of investigation he gave us some new localizations for the speech functions.

It is only necessary to mention the danger of this sort of deduction to cast serious doubt on the value of Head's second conclusions. It is extremely difficult in a severe craniocerebral injury to believe that the only injury to the brain exists exactly under the spot where the cranial defect is. One must understand that this is not the kind of material necessary to help us in understanding this problem.

In Dr. Dercum's case, there is a possible explanation of the situation, based on two points. The anterior portion of the insula, as Dr. Mills has said, is closely connected with Broca's area by association bundles. The architectonic studies of Broadman show further this intimate relationship. The posterior portion of the insula is connected with the primary auditory speech area and with the lenticular nucleus, and a severe injury such as that which occurred in Dr. Dercum's case would produce in effect a rather complete aphasia. This explanation, together with the conception of von Monakow of nerve shock diaschisis, would account for the rather complete anarthria in this case.

The question of the lenticular nucleus in connection with aphasia was dealt with by Mingazzini eighteen years ago. Von Monakow reviewed his work and objected strenuously to tying up the lenticular nucleus with aphasia, because it seems hardly likely from the phylogenetic standpoint that the lenticular nucleus has anything to do with speech. Since then Mingazzini's work has been gone over again, and it was found that in nearly all his cases the lesion had involved not only the posterior part of the putamen where he localized speech on the left side, but also the internal capsule. There were also lesions in other parts of the brain, and perhaps Mingazzini unnecessarily stressed the lesions which he found in the lenticular nucleus and was confusing dysarthrias and anarthrias with aphasia.

It has always seemed to me that aside from the conception of a center for the emissive side of speech, for the regulation and coordination of all the musculature involved in actual utterance, the attempt at localization of speech in any other area is a rather dangerous situation. An intensive review of the literature will show that all of these more or less conventional or set conceptions of speech can be readily upset on the basis of the work of those who report the cases on which their particular conceptions are based.

Dr. M. Allen Starr, New York: This discussion reminds me of discussions which we used to have in 1884. It was a question then whether there was such a thing as sensory versus motor aphasia.

I dislike to have Dr. Dercum ascribe to Déjerine all the distinctions between motor and sensory aphasia and various forms of sensory aphasia. Déjerine's observations were published in 1901. In 1882, in Charcot's laboratory and hospital at Salpêtrière I saw cases of absolutely single alexia with a power of speech, a power of recognition of language, a power of expression of language in speech, a power of expression of language in writing, in persons who could not read and could not recognize words.

There was a case of pure alexia that Charcot used to demonstrate once in three months in his clinic, and oftener in his wards. According to Dr. Dercum, Déjerine says it does not occur, but I have seen a similar case of pure alexia without agraphia in my own practice, because the man could write sentences which he subsequently could not read, and yet he had no defect of speech. I think that a single, well studied case overthrows a great deal of argument and also of hypothesis.

Dr. Bernard Sachs, New York: I should like to take a position midway between the one that Dr. Dercum assumes and that taken by Dr. Mills. It is not surprising that we have been studying this subject for more than forty years. As a result of our thinking and practical experience, I am certain that none of us feels that he can entirely disregard the subdivision that is so familiar, namely, motor and sensory aphasia, and I fully agree with Dr. Starr that there are undoubtedly cases that call our attention to the need of a cortical localization and the determination of cortical centers.

In addition to the cortical centers we have learned that we must regard speech not only as a matter of centers, but especially as a matter of association. It is natural that speech disturbances will occur with lesions in many different parts of the brain. As the association tracts pass not only through the white matter, but also in the vicinity of the ganglia, the internal capsule, etc., I am not astonished that there may be cases such as Dr. Dercum described, in which one lesion may have been responsible for the special form of aphasia that he has observed. I have been trying to be in sympathy with the view of

Pierre Marie, and yet I feel that speech is not so much a question of intelligence as it is one of intellectual processes, and intellectual processes cannot be carried on without engaging a very large part and different parts of the brain.

I could not agree to the idea of regarding speech as a reflex mechanism. Speech, even when we are not conscious of it, requires making an effort, and although every one thinks that he speaks rather glibly, nevertheless it is an effort.

I had a rather sad illustration of "effort" in the case of a medical man, whose very first symptoms proved that although the examiner was not able to appreciate the effort made, the patient felt it. The first symptoms, which was the effort needed to produce speech, appeared two months before any definite signs of the lesion. There was no sign of speech disturbance, so that the examiners were inclined to regard his expression that there was difficulty in his speech as a hypochondriac notion. However, the patient from the beginning of his trouble felt the mechanism of speech or the difficulty of pushing through the effort and stimuli that were necessary in order to bring about speech. It seems that a case like that proves that if we regard speech as a purely reflex mechanism, there is a more or less conscious effort present, and I cannot place speech on the same level with other reflex mechanisms.

From a purely practical point of view, I think those who recall what has been done on the intelligence side of speech should not forget the most marvelous essays that have been written on speech by Hughlings Jackson. While we may learn to appreciate the exact rôle which intelligence plays in speech, I think we shall continue to speak of the centers of motor and sensory aphasia, especially the latter.

DR. BRONISLAW ONUF, Rutherford, N. J.: The late Dr. Joseph Frankel and myself made a careful analysis of 103 cases of aphasia some years ago. They were all cases that came to necropsy, so that we have the anatomic findings to guide us. The results which this analysis has produced have been unsatisfactory; they have been more negative than positive. I think that perhaps we were too critical in the requirements we made for a case that would stand criticism.

We reasoned that in every case of focal lesion there would be a period of shock, of reaction, and inflammatory edema near the focus. We perhaps arbitrarily gave an allowance of two months before we assumed that the focal symptoms which the case presented were permanent ones due really only to the focal lesion.

On this basis, we found that of the 103 cases there were only five with circumscribed lesions of the base of the third frontal convolution, and in not one of those type cases were there any permanent symptoms. None of them presented aphasia which lasted as long as two months. If we are going to judge from this result, we should have assumed that the third frontal convolution really had nothing to do with aphasia. However, I think that this conclusion would be wrong. I would remind you of the areas of innervations of muscle and skin, how we never have a supply by one particular center, but that we always have an overlapping of areas. I think that there is not only no definite area which is confined to the function of speech, but that we have a number of them.

In the first place, we have of course the left and right hemispheres. In that respect we found that there was a cooperative scheme as in many cases a lesion of the right hemisphere would cause temporary symptoms of aphasia, but that after subsidence of these symptoms a subsequently occurring lesion

of the left side would make the aphasia permanent. Sometimes it would be the reverse, that is, aphasia would be produced by a left hemisphere lesion, but would not become complete until a symmetrical lesion on the right side occurred. I think that there may be a sort of parallelism with what Dr. Meltzer once described in a paper as the "safety factors of the organism," that there is always additional stock present that can be drawn on when the usual supply is exhausted.

All these factors must be considered, which makes this subject extremely difficult. I think any conclusions must be drawn carefully, and it is most difficult to reach a conclusion on a case which is of recent origin, because the symptoms of shock cannot be excluded.

Dr. Dercum, Philadelphia: I read my paper hurriedly and in an abbreviated form because of the time that would have been required to present it in full, and in a sense it was misunderstood. I made no attack on the current theories of cerebral localization. I have merely placed a case on record. In the explanation which I advanced, I emphasized the fact that the associations concerned in emissive speech are fixed associations. The innervations which are concerned in these associations are exceedingly complex; they include the innervations of the numerous muscles concerned in respiration, the innervation of the complex muscles of the larynx, of the muscles of the palate, of the tongue, of the buccal cavity and of the lips. These associations are fixed associations; they are not cortical, but belong to the striatum. Originally, the associations are cortical. They are at first consciously performed and with effort, as any one can testify who has acquired a foreign language in adult life. Soon, however, they become fixed associations and the complex coordinated movements required are unconsciously performed. Ordinarily, we do not in speaking stop to think even of the words.

All of the voluntary muscles, and those concerned in speech are of course included, have a double innervation, first that derived from the motor area of the cortex, and second, that derived from the striatum. Both innervations are operative the moment that any movement is made. The cortex deals primarily with variable, adjustable, constantly changing associations. It is possible that fixed associations may be established in the cortex, but one of the things that we have definitely learned about the striatum is that it is especially concerned with automatic associated movements, and further that these associated movements are fixed. It does no violence to the facts in our possession to assume that the striatum plays a definite rôle in all fixed associated movements, and if so it must also play a rôle in the fixed associated movements concerned in spoken speech. If this be true, the loss of emissive speech in the case I have presented becomes intelligible; an important part of the mechanism concerned has been destroyed. Standing and walking are definitely known to depend on fixed associations in the striatum; this is probably true also of swimming, which, though acquired with conscious effort, soon becomes automatic and subconscious. Why can this not be true of all automatic associated movements? Finally, how can we exclude the striatum from this rôle, especially as the striatum has in the vertebrate series steadily increased in size and complexity until it has reached its fullest development in man? We should remember that the neostriatum has grown in size and complexity with the cortex.

I should add that I have seen one case of aphasia in which the man was unable to talk, although he could read and had a full language comprehension. I have placed the case on record; it was not one of hysteria but of brain

syphilis, and the symptom rapidly disappeared on specific medication. I regarded the case as one of Déjerine's cases of "pure or subcortical motor aphasia," and as one of Marie's cases of "anarthria," due likewise to a subcortical lesion, one involving the putamen. What is the difference?

I should like to ask Dr. Starr, whether necropsy was performed in Charcot's case?

DR. M. ALLEN STARR: I do not know.

DR. DERCUM: It may have been a case of some functional disturbance and not organic in character.

MUCIN-LIKE BODIES IN THE CENTRAL NERVOUS SYSTEM IN EPIDEMIC ENCEPHALITIS *

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Few references to the presence of mucin or mucin-like bodies in the central nervous system have been found. Grynfeldt [1] has recently described mucin deposits in the brains in cases of epidemic encephalitis, Wilson's disease, parkinsonian syndromes, anemias and typhoid fever. The neuropathologic texts of Spielmeyer,[2] Buzzard and Greenfield,[3] and Bertrand [4] make no comment on this subject.

The staining qualities of mucin are well known. Lee [5] makes the general statement, quoting Hoyer, that "the mucin of mucus cells and goblet cells stain with basic tar dyes and with alum haematoxylin, but not with acid tar dyes." Lee makes no mention of the presence of mucin in the nervous system. Langeron [6] states, in discussing mucin, that it is stained red with thionin, methyl violet, toluidin blue and polychrome blue. Both Langeron and Lee state that Mayer's mucicarmin mixture is specific for mucin. This mixture will also stain other substances and structures, such as hyaline cartilage, Wharton's jelly and Ehrlich's Mastzellen (Lee).

Metachromatic substances both within and without the cells of the nervous system are commonly found. Such bodies having special staining qualities were reported by Lhermitte and Radovici [7] in a case of

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* From the laboratory of neuropathology of the University of Paris.

1. Grynfeldt: L'oedeme de Lèri est en realité une dègènerescence muqueuse des cellules interstitielles, Soc. des Sc. méd. et biologiques de Montpellier et du Langedoc Mediterranéen, Dec. 21, 1923; Du rôle des mucocytes dans certains processus encephalitiques, Ibid., Feb. 4, 1924.

2. Spielmeyer: Histopathologie des nervensystems, 1922.

3. Buzzard and Greenfield: Pathology of the Nervous System, 1922.

4. Bertrand: Les processus de désintegrations nerveuses, 1923.

5. Lee: The Microtomist's Vade-Mecum, Ed. 8, 1921.

6. Langeron: Précis de microscopie, 1921.

7. Lhermitte and Radovici: Étude sur la dégeneration basophile metachromatique, des fibers et des cellules du cerveau et de la moëlle épinière dans l'encéphalite épidémique, Compt. rend. Soc. de biol. **84**:931 (May 21) 1921.

epidemic encephalitis. They gave metachromatic reactions with basic dyes, but were not visible with Nissl's methods and did not stain with safranin, eosin, trypan red, sudan III or Mann's mixture (methyl blue-eosin). They stained well with Casamajor's and Bonfiglio's methods. They were insoluble in alcohol, ether and chloroform. Lhermitte and Radovici were unable to find reference to bodies having these staining reactions. No stain for mucin was used, and the possibility that the bodies which they found were mucin was not mentioned.

The bodies found by Lhermitte and Radovici were mainly in the white matter of the brain. A few were scattered through the gray matter. In the cord, they were found principally in the columns of Burdach and Gowers and the crossed pyramidal. The bodies found by Grynfeldt [8] were present in the white matter of the brain, the corpus callosum, the corona radiata and internal capsule.

In the brain of a patient with chronic epidemic encephalitis of the parkinsonian variety, dying of pulmonary tuberculosis, we have found the same kind of bodies or deposits. They lay in the neuroglial meshes, had no cytologic structure and were not found within neuroglia or nerve cells. They were present in the white matter of the cerebellum adjacent to the dentate nucleus. Their form was spherical, with irregular edges on high magnification. Their diameter varied from 20 to 50 microns. They were clearly not artefacts, nor due to the presence of celloidin masses. Grynfeldt [8] has called these bodies mucocytes. With this we cannot agree, as they have no cell structure. What their origin is, we cannot say.

The staining qualities of these deposits are indicated in the table, their appearance in Figures 1 and 2. It will be seen that they stain with basic dyes, but not with acid dyes. With basic dyes, metachromatic reactions were always found, except with mucicarmin. We disagree with the findings of Lhermitte and Radovici [7] in regard to safranin, since we have found that it shows a yellow metachromatic staining, faint but definite. Langeron [6] mentions this reaction with mucin. The mucicarmin reaction is striking—a deep rose color.

With hematoxylin, the bodies stained a beautiful light blue, like that seen in the mucus cells of the gastro-intestinal tract.

With Nissl's method, as has been observed by Lhermitte and Radovici,[7] these bodies remain unstained. This was the result when the differentiation of the staining with toluidin blue was carried to the proper point. Examination of the section before this point was reached, however, showed the bodies to have a pink color like that obtained with

8. Grynfeldt: Soc. des Sc. méd. et biologiques de Montpellier et du Langedoc Mediterranéen, Feb. 4, 1924.

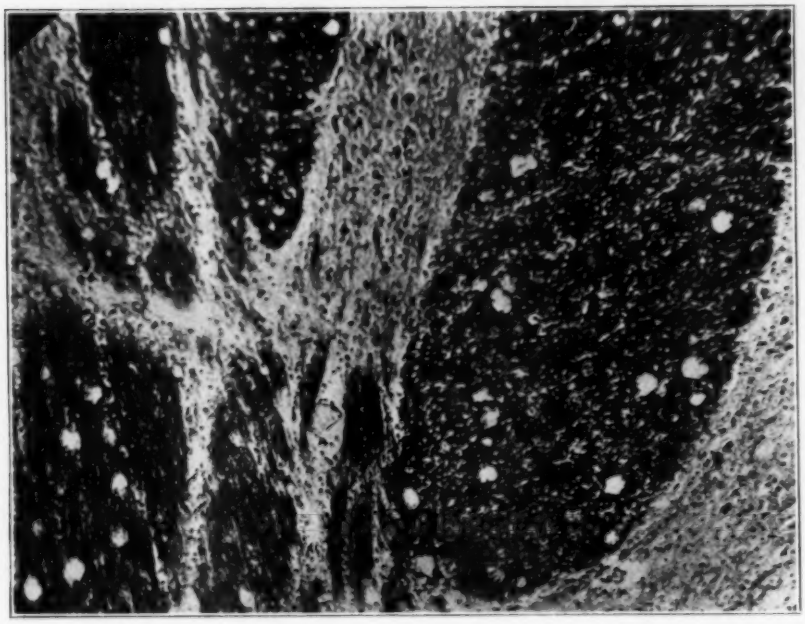

Fig. 1.—Section of the pons (20 microns). Stained for myelin sheaths by the method of Loyez. Note the absence of staining of the deposits. × 120. Compare with Figure 2.

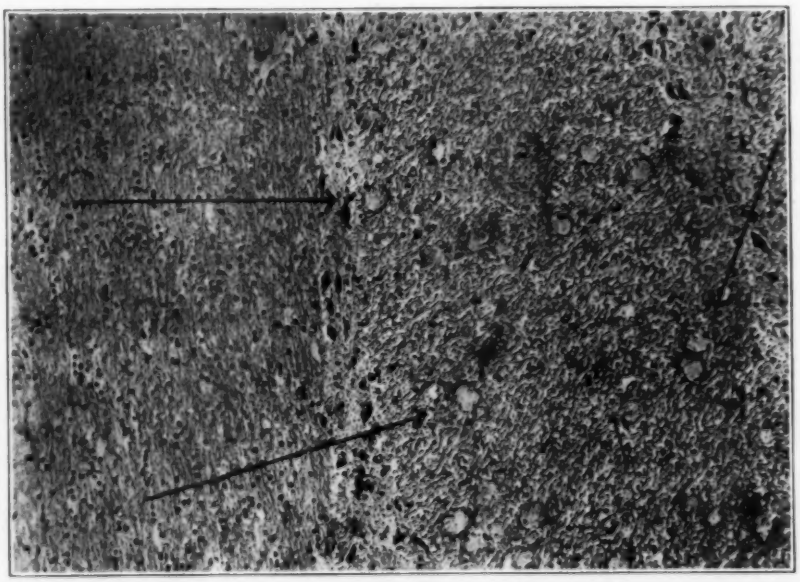

Fig. 2.—Section of the pons (20 microns). Hematoxylin and van Gieson stains. Note the deposits which were light blue in contrast to the deep purple of the elements stained with hematoxylin. × 120.

toluidin blue alone (see table). In order to ascertain whether the anilin oil used in differentiating had dissolved the bodies or the toluidin blue, a section was placed in anilin oil for twenty-four hours and then stained with mucicarmin. The bodies stained well, showing that the action of the anilin oil was on the toluidin blue.

TABLE 1.—*Results of Various Stains*

A. Basic Dyes		*Remarks*
1. Toluidin blue	First violet, then rose after differentiation is sufficient.	Metachromatic.
2. Polychrome methylene blue (Unna)	Rose	Metachromatic
3. Thionin	Rose	Metachromatic
4. Methyl violet	Rose	Metachromatic
5. Safranin	Yellow	Metachromatic
6. Neutral red	Red	
7. Mucicarmin	Red	
B. Acid Dyes		
8. Lyon blue	No color	See No. 25
9. Methyl blue	No color	See No. 24
10. Anilin blue	No color	See No. 26
11. Light green	No color	See text; method of Prenant
12. Magenta S.	No color	
13. Eosin (water solution)	No color	See Nos. 22, 24, 25
14. Picrofuchsin	No color	See No. 23
15. Acid fuchsin	No color	See No. 23
16. Orange G	No color	See No. 26
17. Metanil yellow	No color	See No. 28
C. Natural Dyes		
18. Hematoxylin	Light blue	
D. Methods		
19. Nissl (tol. blue)	No color	Differentiation with anilin oil
20. Loyez (myelin sheaths)	No color	
21. Perl (for iron) counterstain, neutral red	No color except with neutral red	
22. Hematoxylin and eosin	Light blue	
23. Hematoxylin and van Gieson	Light blue	
24. Mann	Light blue	
25. Leroux (9)	Light blue	
26. Mallory	Light blue	
27. Klarfeld—Achùcarro	Black before complete differentiation; then no color	
28. Mucicarmin after hematoxylin and metanil yellow	Red	

A striking result was a faint blue color with the methods of Mallory, Leroux and Mann. The last disagrees with the findings of Lhermitte and Radovici.[7] When the anilin blue (Mallory's method), Lyon blue (Leroux's method) and methyl blue (Mann's method) were used alone, no staining resulted.

In the two latter, eosin is used before or with the blue dye. Its mordant action may be the cause of the different behavior of the blue dyes. It is interesting in this connection that in the method of Prenant (overstaining with eosin, then hematoxylin and finally light green), the mucin stains green.[9] With the acid dye, light green alone it does not stain. The eosin may play a part here too. In Mallory's method, orange G, whose physicochemical action is known to be complicated in relation to other dyes, may be the cause of the staining with anilin blue.

The staining reactions with hematoxylin showed that these deposits were not amyloid, and the reaction to acid fuchsin, that they were not hyalin.

With the Klarfeld-Achùcarro method, these bodies were first stained black (silver), but further differentiation dissolved the silver and left them colorless.

The fact that these bodies or deposits showed all the characteristic reactions of mucin when found elsewhere in the body, leads us to believe that they are chemically either mucin or a closely related substance. Their specific staining with Mayer's mucicarmin lends weight to this opinion.

CLINICAL AND PATHOLOGIC REPORT

A married woman, aged 24, near the end of pregnancy, in 1919, was taken sick with a lethargic form of epidemic encephalitis. Delivery occurred during the lethargy. The child was alive in 1922. The patient developed a typical parkinsonian case about a year after the onset. She was compelled to enter a hospital. A cavity was found at the apex of the left lung. Pulmonary tuberculosis was diagnosed, and the patient died in 1921 of that disease.

At necropsy, lesions of pulmonary tuberculosis were found. The brain seemed normal. The cord was not removed. Sections showed, in addition to the deposits already noted, the typical picture of chronic epidemic encephalitis. The substantia nigra cells were very much involved, but few remained. There was round cell infiltration and capillary dilatation in this region. The cells of the globus pallidus were moderately involved, there being diminution in number and evidence of degeneration. The blood vessels in the midbrain, subthalamic region and region of the basal ganglions showed a surrounding "etat criblé" of typical character. There was no round cell infiltration about the vessels. There was no evidence of degeneration of fiber tracts or of myelin sheaths.

CONCLUSION

Mucin or a closely allied substance having the staining qualities of mucin may appear in the brain in chronic epidemic encephalitis.

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9. Langeron: (Footnote 2) p. 801.

FRACTURE-DISLOCATION OF THE NECK

A METHOD OF TREATMENT *

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NEW YORK

In fracture dislocations of the neck with signs of a cord lesion, it is desirable to relieve the cord of continuing pressure resulting from bony deformity. This may be done either: (1) indirectly by performing a laminectomy, going well above and below the lesion, followed by longitudinal splitting of the dura, i. e., a so-called decompression of the spinal cord, which permits displacement of the cord backward and frees it from the scissor-like compression resulting from the usual bony distortion; (2) directly by manipulation which reduces the dislocation, followed by fixation in normal position.

Many years ago, while on duty at Fordham Hospital, three successive fracture dislocations of the neck were treated by laminectomy and splitting of the dura, the interval between injury and operation varying from one to four days. In each case, when the dura was split over the traumatized area, the cord fractured and extruded damaged fragments. Shortly after operation, the temperature rose sharply, and death occurred within twenty-four hours, with the temperature around 107 F.

It was then suggested that a laminectomy, without splitting the dura, would remove the direct bony compression. However, the brilliant experimental work of Reginald Allen, of Philadelphia, on injuries of the spinal cords of dogs indicated that edema of the cord secondary to contusion was sufficient to cause complete transverse lesion of the cord unless the dura was opened promptly and the cord incised to permit escape of the edematous transudate.

Laminectomy with splitting of the dura had given consistently disastrous results. Laminectomy without opening the dura gave promise of but slight, if any, benefit in return for an open operation, which, under the circumstances, involved serious additional risk to the patient.

A method, therefore, which would eliminate the bony distortion and continuing external pressure on the cord without the added risk of open operation, would favor spontaneous recovery in the cord as far as at present seems possible.

The method herewith presented was first tried in August, 1911, and in two other cases in 1920 and 1922, respectively. It is simple and has

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* Read at the Fiftieth Annual Meeting of the American Neurological Association, Philadelphia, June, 1924.

proved efficient. The principle consists in controlled traction exerted on the head (with counter traction on the lower extremities) until the spasmodically contracted cervical muscles have relaxed sufficiently to permit reduction by bimanual manipulation on the part of the operator. Fixation is maintained by a plaster jacket involving the trunk and neck, the neck portion maintaining extension of the neck by pressure upward against the occiput and inferior maxilla, and downward against the shoulders. The most convenient arrangement is that shown in the accompanying illustrations. The patient is placed supine on a Hawley fracture table, with the trunk from the waist line upward supported on a narrow metal strip padded slightly and ending between the scapulae.

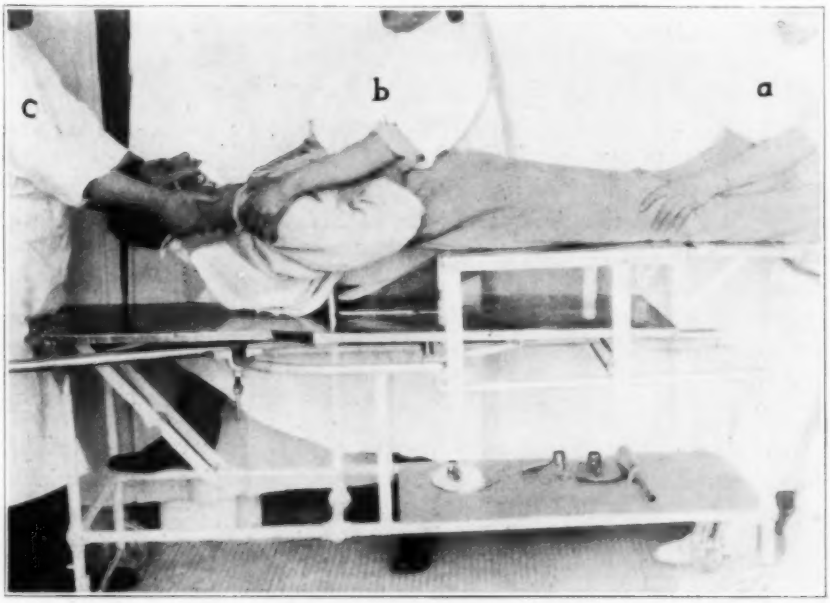

Fig. 1.—Patient on Hawley fracture table with everything ready for reduction. (*a*) counter extension on lower extremities; (*b*) steadying of shoulders to keep patient from slipping off the narrow metal spinal strip; (*c*) operator exerting traction by his body weight while steadying the head and neck with both hands.

A leather contrivance used by orthopedists to suspend patients by the head is placed so as to give traction on the patient's chin and occiput. Through its two suspension rings which lie just above the vertex of the patient's skull a double bight of clothes line is passed, and, continuing around the waist or pelvic girdle of the operator, it is tied so as to keep the patient's vertex only a short distance from the operator, who faces the patient and grasps his neck in the damaged area. Meanwhile, the lower extremities of the patient are bound to the table, or held

by strong assistants, to give counter traction. Another assistant may be necessary to keep the patient's body balanced on the narrow metal strip between his scapulae (Figs. 1, 2 and 3).

Fig. 2.—Details of extension apparatus applied to head, with rope around operator.

Fig. 3.—Details of head end of apparatus.

In two of the cases reported, no anesthetic was used, as there is usually no pain from the manipulation. In the third case, because of painful, clonic spasms in the neck muscles, and nervousness on the part of the patient, general anesthesia was used.

When everything is ready, the operator applies traction on the neck muscles, gradually and increasingly by backing his body away while holding the neck between his two hands, thus maintaining absolute control of the whole procedure. The traction is at first exerted in the axis of that portion of cervical spine ABOVE the injury so as to unlock the articular processes of the damaged vertebrae. After traction for a period varying with the strength and degree of spasm of the neck muscles (from five to ten minutes), the neck will be felt to elongate gradually, the bones to unlock, and then the head and upper spine are allowed to sag gently downward while still under traction until reduction is accomplished, the operator's hands assisting by propping the lower segment of the cervical spine and manipulating the upper segment gently with the thumbs, if necessary, to complete the reduction.

Reduction is indicated: (*a*) sometimes by the patient who feels the bones slide into place, with immediate relief of previous discomforts; (*b*) by finding the spinous processes in proper alinement and spacing; (*c*) by a plate taken by a portable machine and developed while the patient is retained on the table.

As soon as the reduction is verified, a good orthopedist should apply a well padded plaster of Paris jacket (previously described) while the operator maintains the head and neck in correct position. This jacket should insure moderate extension of the neck between shoulders and occiput and chin, with slight backward flexion of the head and upper part of the neck to check any tendency to recurrence, or to undue erosion of the anterior portions of the damaged vertebral bodies. This jacket is kept on for many weeks, until the damaged region has had plenty of time for primary repair. It may then be replaced by a leather jacket and neck piece properly reinforced by steel strips. Such support must be maintained until good bony union has occurred between the damaged vertebrae, which apparently requires about a year.

REPORT OF CASES

Case 1.—W. B. L., a youth, aged 17, on Aug. 12, 1911, while surf bathing, dove through a roller and landed on his head. There was immediate paralysis. He was carried out by his companions. Primarily, there was complete loss of cord function below the fifth cervical segment. During the first week, there was some return of sensation, slight motion in the big toes, but no sphincteric control. A roentgenogram showed displacement of the fifth cervical vertebra forward on the sixth cervical vertebra about 1 cm.

On August 19, he was first seen in consultation, and the foregoing method was applied. Thereafter he made steady and fairly rapid recovery. He was not seen again until one year later, August, 1912, when he was driving his own runabout, had begun to dance again and was well except for a moderately troublesome residual spasticity in the lower extremities, and a sharply circumscribed hypesthesia of the tip of the right index finger. Sphincteric control had been recovered during the first two months. In 1920, nine years after

injury, there was still a slight degree of spasticity of the lower extremities, which, however, caused no disturbance in gait except on first rising in the morning. The tip of the right index finger had become normal in sensation.

CASE 2.—J. F., a man, aged 27, was always well, but of a nervous, irritable temperament, exaggerated by his army experience. For years he had had recurrent dislocations of the left shoulder, and of both patellae, following injuries. On Feb. 23, 1920, while wrestling, he felt a crack in the middle of his neck, and fell limp to the floor. He was lifted into bed. After recovering from the immediate shock, he noted marked weakness in both upper extremities, although their range of motion seemed normal. There was no paralysis of the sphincters nor of the lower extremities, and he noticed no interference with breathing other than that he could not get force enough to blow his nose satisfactorily. Rotation of the head or attempts to raise it from the bed caused pain in the neck. For the first forty-eight hours, there was increasing weakness in the upper extremities until only the fingers could be moved. He then began to recover gradually.

On February 28, when he was first seen in consultation, he was lying supine, with the arms abducted 90 degrees, elbows flexed, forearms pronated and hands lying on his chest (the typical attitude for injury of the sixth cord segment). The left shoulder, elbow and wrist joints could be moved normally and with moderate strength. The left digits could be flexed and extended, but with almost no power. The right shoulder and elbow joints showed marked loss of power, but the digits moved with almost normal freedom and power. On the right upper extremity, there was diminished sensibility to pin prick in the musculospiral field from the upper arm to the hand. On the left side, the sensory disturbance was less marked and extended from the elbow down the forearm and hand in the distribution of the radial nerve. Elsewhere sensation to pin prick was normal. Thermal sensibility was normal down to the level of the fourth dorsal in back and the second dorsal in front. Below these levels both hot and cold were interpreted as hot. The reflexes of the upper extremities, abdomen and cremaster were absent bilaterally. The knee reflexes were exaggerated, but equally so, and the ankle reflexes were present bilaterally. There was no ankle clonus on the right, but an exhaustive clonus on the left. Babinski's sign was absent on both sides, but the Oppenheim sign was present.

There was no apparent loss of power in the lower extremities. Bending the head forward and rotating the head and neck to either side caused marked pain. There was marked tenderness to pressure over the transverse and spinous processes of the lower cervical vertebrae, and marked prominence of the spinous process of one of the lower ones.

Roentgenograms showed considerable dislocation forward of the fifth on the sixth cervical vertebra (Fig. 4). The findings indicated that the fracture dislocation had caused injury to the peripheral nerves running to the upper extremities and slight injury to the cord itself as shown by the changes in the reflexes and in thermal sensibility. The absence of motor paralysis below the lesion would show that cord injury had been slight.

The indications for reduction were: the marked dislocation, the damage to the plexus roots and the danger of transverse myelitis resulting from pressure on the cord by the sharp edges of the displaced vertebrae.

March 3, 1920 (eight days after the injury), the procedure above outlined was carried out without any anesthetic. As soon as traction was started, the patient said his neck was more comfortable, and after about five minutes, he said that he felt something slip in his neck, and he felt perfectly comfortable.

He could rotate his head well to either side without pain. Examination revealed the spinous processes again in normal relation, and the plaster jacket was applied by an orthopedist. Roentgenograms taken, March 6, 1920, showed a good reduction (Fig. 5). After about two weeks, a reinforced leather collar was substituted for the plaster jacket, and he returned home.

On March 23, learning that he had removed the leather collar for shaving, bathing, etc., further pictures were taken, which showed an incomplete recur-

Figure 4

Figure 5

Fig. 4 (Case 2).—Before reduction. Dislocation of the fifth cervical forward on the sixth cervical vertebra. Note locking of articular processes and that extension at first must be forward to unlock them with minimum force and traction.

Fig. 5.—Reduction first time. Note that intervertebral disk is diminished in thickness and articular processes are not quite seated home.

rence of the deformity. On March 26, the reduction was repeated and a plaster jacket again applied. Following this reduction, a steel spinal brace with a jury mast for extension of the neck and support of the head was made and substituted for the plaster jacket. On June 16, 1920, there had been complete recovery of motion, sensation and power except for slight limitation of the right arm in moving backward and upward. However, lately, there had been slightly

less freedom in lateral rotation of the head and neck. On June 19, 1920, a roentgenogram showed partial recurrence. It was decided to attempt open reduction and fixation, and accordingly, on June 24, 1920, under morphin-procain-epinephrin anesthesia, a posterior median incision was made, the spinous and articular processes and laminae of the fifth and sixth cervical vertebrae being clearly exposed. The surprising feature about the dissection was that after the long interval since injury (four months) there was no appearance of healing in any of the tissues.

Figure 6

Figure 7

Fig. 6.—One year after the primary reduction, two recurrences of deformity having occurred and attempt at fixation by open operation having been made. Note the deformity, and that there is no sharp angulation of the spinal cord, that the intervertebral disk has almost disappeared anteriorly, and that bony union has occurred between the vertebral bodies anteriorly.

Fig. 7.—Four years after reduction showing ossification between bodies and articular processes of the fifth and sixth vertebrae. Note the extension downward of the anterior lip of the fifth cervical. Intervertebral disk almost gone.

Except that the extravasated blood had been absorbed, the field looked like a fresh injury. This appearance associated with the recurrent dislocations of his shoulder and patellae indicated a serious defect in reparative power. Even so, by no permissible use of force and manipulation could

the deformity be reduced more than about 25 per cent., probably because of the mechanical locking of the front edges of the two vertebrae. Finally, two heavy silk ligatures were passed about the spinous processes of the two vertebrae and tied firmly when the best approximation possible had been obtained. The wound was closed by layer sutures. The jury-mast brace was immediately replaced. Primary union resulted.

At intervals of two months, roentgenograms were taken to ascertain whether bony union of the two vertebrae was occurring. Finally, on March 17, 1921, slightly more than a year after injury, bony union between the bodies and also between the articular processes was so well advanced (Fig. 6) that the brace was discarded. His recovery was complete. On March 17, 1924, examination showed complete recovery of motion, sensation and power. All reflexes were

Fig. 8.—Anterior view of Figure 7.

normal except for a slight exaggeration of the knee reflexes. There was no sign of spasticity in his gait. He has been attending to his business since 1921 (Figs. 7 and 8 show good bony union).

Case 3.—L. Z., a woman, aged 35, on Oct. 25, 1922, in an automobile collision, was throw out, landing on the pavement on the back of the head. Examination by Dr. E. H. Pool revealed a symptomatic complete transverse lesion of the cord at the sixth cervical segment. There were great pain and frequent spasm of the muscles of the neck, which persisted up to the time of reduction. On October 26, twelve hours after the injury, a neurologic examination by Dr. Foster Kennedy revealed: a transverse lesion of the cord at the level of the sixth cervical segment, tenderness over the sixth cervical vertebra, and motion absent in the legs and trunk. In the arms, the right and left biceps and deltoid were intact in power; the triceps was crippled on both sides, and the hand and forearm muscles were paralyzed on both sides. All deep reflexes were abolished; the left plantar reflex was absent, and the right slow in flexion (a pathologic sign). Pain (superficial) and touch were abolished up to the level

of the sixth cervical vertebra. Appreciation of temperature was lost one and one-half inches higher than that of touch or pain. The patient vaguely felt deep pain in the legs, and in movement of the toe joints.

There was no complete crush of the cord; it was not possible to be sure that she did not have a hematomyelia, but Dr. Kennedy believed that her lesion was a bony crush fracture on the fifth and sixth cervical vertebrae. He advised waiting for roentgenograms.

There was complete loss of sphincteric control.

My own examination showed the region of the fourth to the seventh cervical to be extremely tender, both front and back but mostly over the fifth and sixth cervical vertebrae, where there was some deformity which could not be accurately determined because of tenderness. There was a fulness of the front of the spine in the region of the sixth cervical. Breathing was entirely diaphragmatic and oral because of old nasal difficulty.

Figure 9

Figure 10

Fig. 9 (Case 3).—Before reduction. Note extreme forward displacement of the fifth cervical vertebra and locking of articular processes.

Fig. 10.—Taken on the table immediately after reduction, showing complete reduction.

Any movements of the head, neck or even the body caused extreme pain. A roentgenogram (with portable, Fig. 9) showed extreme dislocation forward of the fifth cervical on the sixth, with some crushing of the body and articular processes on one side.

On October 27, the neck was stretched and complete realinement secured. The articular processes did not seat entirely home, but they were unlocked, replaced and seated about two-thirds home. The alinement of the bodies laterally and anteroposteriorly was perfect. A lateral roentgenogram was taken while the patient was on the table (Fig. 10). A plaster cast involving the chest, neck, jaw and occiput was applied with the neck slightly hyperextended.

From the moment of reduction, her pains and muscle spasms disappeared, and she could move her head laterally and rotate it slightly in her cast without discomfort. Four days after the reduction, another picture was taken in order to make sure of the position, and there was perfect alinement, even the articular processes having seated home into their normal positions (Fig. 11). She made slow but uninterrupted improvement.

The day following the reduction, the patient had decidedly more strength in those muscles of the upper extremities which she had been able to use before the reduction had occurred. On the sixth day, following reduction, there occurred definite movement in the toes of the right foot and slight movement in the great toe of the left foot, although this was open to question. There was also a shadow of movement in the thumb on the left hand. On the twenty-

Fig. 11.—Three days after reduction, showing normal alinement.

second day, the patient flexed the third and fourth digits of the right hand. On the twenty-eighth day, she moved the second and third digits of the left hand. Movement of the fingers of the right hand were very much more definite and stronger than on the left. There was a slight suspicion of movement in the second and third toes of the left foot.

While at first breathing was entirely diaphragmatic, within three weeks the intercostals seemed to be working again. On the thirtieth day, the patient felt sure that her breathing was almost normal, and one could make out definite excursion of the ribs during respiration. From this time on, movement progressed steadily. She developed sufficient power to raise the left leg off the bed. On the thirty-fifth day, there was definite increase in strength in the thumb, and the first and second fingers of the right hand. She began to use the right hand in various ways, especially for rubbing her nose and attempting to scratch, etc. On the thirty-eighth day, she could move the left big toe

definitely, and also raise the knee slightly from the bed. On the forty-fifth day, another roentgenogram was taken, and the spine was still in perfect position. On the forty-ninth day, she complained of cramps in the bladder and passed some urine spontaneously. On the fiftieth day, she voided a large amount of urine voluntarily. From this time on, she was able to control the bladder. There was also steady, definite but slow improvement in all of her muscles in both the upper and lower extremities. On the fifty-fifth day, the cast was split and removed long enough to take an impression for the making of a leather collar and support. Afterward, the plaster cast was replaced and held

Fig. 12.—Sixteen months after reduction with the use of constant extension apparatus. Note the absorption of the anterior portion of intervertebral disk with tilting forward of upper spine and consequent slight angulation. Note also the well advanced ossification between the two vertebrae.

with adhesive plaster. On January 5, the leather collar and jacket was put on. After a few minor adjustments under the chin and back of the head, it was comfortable and seemed to hold the neck and head in proper position. It was laced up on both sides of the neck and over the shoulders.

She went home on January 28, three months after the accident, feeling well in every way. She was able to stand without assistance the day before she left the hospital, but she felt as though her legs were not really under control.

Dr. Kennedy reported, March 23, 1923:

"Power has returned well in the right upper extremity, but the left upper extremity still shows 'main en griffe.' There is spastic weakness of the left lower extremity. Control of the bladder has returned; but not of the rectal sphincter. Abdominal, right and left reflexes are absent. The knee and ankle reflexes are three plus. The plantar reflexes are present on both sides. Sensory Findings: There is loss of superficial pain over the right side of the trunk and the right lower extremity; also in the dorsal root and lower cervical root areas on the right upper extremity, and to a less degree on the left upper

Fig. 13.—Anteroposterior view of Figure 12.

extremity. Temperature is also affected, but touch is normal. The sense of position is normal. There are no new signs of compression, but subjective numbness, recently felt, is a compression symptom and must indicate invasion of the spinal canal space (by a slipped body?). The Brown-Séquard syndrome still present might result from original compression and need not indicate persistent compression at this time on the left cervical cord."

On Dec. 17, 1923, she walked very much like a person with spastic hemiplegia, and leaned on her friend for support. Later, she walked a distance of 8 or 10 feet by herself, and did very well. My findings were: The left lower extremity was markedly spastic and had a tendency to rotate inward. She was feeling pretty well in general and was working ten or twelve hours a day. She still wore her leather collar. With her right upper extremity she could do practically

a full range of motion and had almost a normal degree of strength. With the left upper extremity, she could abduct the arm to about a right angle, flex the elbow about 40 degrees and supinate the forearm so as to bring the palm of the left hand on the occipital region. She still suffered from uncontrollable spasmodic contractions of her left fingers, hand and wrist. She was able to approximate the thumb and index finger with considerable strength so that she could pick up and hold things fairly well. She could extend the other three digits of the left hand almost to a straight line, and could also flex fairly well so as to grip with considerable strength any object about 3 or 4 cm. in diameter. There was loss of pin prick in the musculospiral distribution on the right, especially the lower radial, but she could feel pin prick indistinctly in the ulnar and median areas. In the left upper extremity, exactly the reverse was obtained. She was able to feel pin prick in the musculospiral distribution, but neither in the ulnar nor median distribution. The reflexes of both upper extremities were exaggerated; the left more than the right. Tapping the biceps tendon on both sides caused flexion of the wrist and fingers as well as slight flexion of the elbow. Tapping the deltoid of either side also caused flexion of slight degree of the fingers and wrist. In the lower extremities, there was marked spastic paralysis on the left. The knee and ankle reflexes were markedly exaggerated. There was no ankle clonus. She could flex the left knee voluntarily to 90 degrees, when sitting, and could extend it completely at will. She could also rotate the extremity as a whole inward and less actively outward. In the right lower extremity, she had rather free motion in all joints. The knee reflex was markedly exaggerated, also the ankle reflex, and there was a well sustained ankle clonus. Tapping above the patella on a finger lying transversely, there was a marked jump of the quadriceps extensor. There had been definite and apparently continuing improvement in the left hand, also in the left lower extremity. Her chief difficulty lay in the effort to balance herself while trying to walk.

Feb. 29, 1924: A roentgenogram showed solid bony union of the fifth and sixth cervical, and the leather support was discarded (Figs. 12 and 13).

March 26, 1924: Now, seventeen months after injury, she could walk much more steadily without support, although the left lower extremity was spastic. She had full range of motion and strength throughout the right upper extremity. The left upper extremity showed almost normal range in the shoulder and elbow joints, and considerable power. Supination was slightly limited. The digits still showed spastic contraction, but were gradually improving.

COMMENT

While three cases are, of course, not sufficient to justify any generalizations, the facts brought out suggest several matters worthy of discussion. Discussion will be more to the point if the causes of symptoms, both immediate and remote, are systematically reviewed, and the logical principles of treatment taken up.

Causes of Symptoms:

Fracture Dislocation:

Results:

1. Immediate:

(*a*) Cord: Compression, contusion, laceration, hemorrhage, gross or punctate; rapidly followed by edema. More or less degeneration of cord substance occurs according to the severity and duration of the factors involved.

(*b*) Nerves: Compression, laceration, contusion as they pass through the foramina between the vertebrae involved.

(*c*) Bones and Disk: Contusion, laceration, comminution, damaged blood supply.

2. Remote:

(*a*) Cord: Transverse pressure myelitis caused by edges of dislocated bones if reduction has not been accomplished. Also, more or less degeneration of cord substance according to the degree of "immediate" injury.

(*b*) Nerves: Delayed regeneration according to persistence of compression by bony deformity, or formation of cicatricial tissue as the result of contusion, laceration, etc.

(*c*) Bones and Disk: Softening, erosion and absorption resulting from damaged nutrition. If extreme, kyphosis with increasing angulation and compression of the cord will occur.

The fundamental aim of treatment must be to minimize both the immediate and remote results of the injury to the spinal cord and nerves. Obviously, one of the most important steps must be the earliest possible neurologic and roentgenographic study followed by reduction of the deformity, with fixation in normal position. This removes *continuing* trauma from the nerve structures. There is still left to be combated the contusion, laceration and secondary edema of the nerve structures resulting from the primary injury. The dura is a firm, inelastic tube which presents unyielding resistance to the swelling cord, and if this swelling be great enough and sufficiently prolonged, there will be degeneration of the cord much increased over that due merely to the primary impact. For relief of this edema, consideration must be given to open operation, which, as far as reduction of deformity is concerned, cannot compare with the simple and efficient method above described.

Allen's experimental work on dogs showed that cords, traumatized beyond recovery under ordinary circumstances, could be salvaged to a considerable degree if the dura were freely opened and the cord itself incised posteriorly in the damaged area to permit easy escape of the edematous fluid. He found that satisfactory results depended on splitting the dura and cord within comparatively few hours after the injury. It was after the presentation of his work that the patients in the three cases at Fordham, previously cited, were operated on. It was impossible to operate within the number of hours prescribed, and the disastrous result in each of the cases, and especially the condition of the cord substance as it spurted through the incised dura, led me to believe that the patient's chances were better without open operation. Someone else may, for various reasons, have more success and present sound arguments for operative treatment in selected cases.

Whether the use of hypertonic salt or magnesium sulphate solutions would influence the edema of the cord and also whether cupping or thermal therapy would have any value, remains to be seen.

In all spinal injuries in which the intervertebral disk is lacerated and contused and the involved vertebral bodies are somewhat crushed, there is serious interference with nutrition of the damaged structures, which causes softening and absorption, varying with the degree of injury, and becoming more and more evident with the lapse of months. In the accompanying roentgenograms, this absorption is definitely shown, and it occurred in spite of the constant use of a support to keep the weight of the head off the damaged vertebrae (Figs. 6, 7 and 12). For this reason, it seems to be of prime importance to keep the support on constantly until solid bony union between the two damaged vertebrae has occurred, so as to minimize the ultimate deformity. This has required about one year in Cases 2 and 3. Meanwhile physical therapeutics and muscle training must be systematically used.

SUMMARY

Fracture dislocations of the cervical vertebrae should be reduced as soon as possible after careful examination has determined the condition. The best method at present is by manipulation.

The original technic herein described has the advantages of simplicity, of complete control of the injured structures, of avoidance of additional injury and of facility in checking up the success of the reduction by means of a roentgenogram.

The patient must wear a support continuously, day and night, until solid bony union has occurred between the damaged vertebrae.

Physiotherapy and muscle training must be used to obtain the best results.

The same principles may well apply to fracture dislocations in lower portions of the spine, and a modification of this technic would assist materially in improving results.

A STUDY OF THE HISTOPATHOLOGY OF AMAUROTIC FAMILY IDIOCY (INFANTILE TYPE OF TAY-SACHS) *

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The term "amaurotic family idiocy" comprises a large group of disorders in which there are more or less definite pathologic changes. Here belong the infantile type of Tay [1]-Sachs,[2] the late infantile variety described by Jansky,[3] Schob,[4] but especially well studied by Bielschowsky [5] and the juvenile form of Vogt [6] and Spielmeyer.[7] Ichikawa [8] accepts six varieties in which, he states, the changes, especially the retinal, as well as the clinical features are practically alike. Vogt, Spielmeyer and Bielschowsky also class with amaurotic family idiocy

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* Presented at the Fiftieth Annual Meeting of the American Neurological Association, June, 1924.

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cases of exquisitely hereditary and familial cerebral disorders recorded by Pelizaeus,[9] Higier,[10] Merzbacher[11] and one lately reported by Wolpert.[12] However, like many other cases of blind idiocy, such as Winkler's,[13] these have little in common, either clinically or anatomically, with the three well studied types mentioned above. Possessing common clinical features—blindness and progressive mental deterioration—these three groups are generally considered varieties of one disease, for they also show a number of histopathologic changes fundamentally alike.

The main pathologic problem pertains to the nature of the disease process; whether it is an inflammation, degeneration or a congenital anomaly; whether it is a diffuse or focal lesion: that is to say, whether the entire central nervous system is involved or only certain portions.

It is generally admitted that inflammation is absent, the disease being regarded either as a degenerative or a developmental anomaly. Sachs,[14] the principal discoverer of this disease, was first inclined to consider it a congenital, genetic morbid condition with involvement of "the highest nerve elements" and a defective "Anlage" of the entire central nervous system, the degenerative factor being nonessential.

On the other hand, Schaffer[15] believed amaurotic family idiocy to be a degenerative process affecting a rather normally "predisposed nervous system, important developmental anomalies hardly ever occurring." In one of his subsequent numerous publications on this subject, Schaffer[16] somewhat modified his views maintaining that degenera-

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11. Merzbacher, L.: Eine eigenartige familiär-hereditäre Erkrankungsform (Aplasia axialis extracorticalis congenita), Ztschr. f. d. ges. Neurol. u. Psychiat. **3**:1, 1910.

12. Wolpert, I.: Klin. Beitrag zur progressiven familiären cerebralen Diplegie, Ztschr. f. d. ges. Neurol. u. Psychiat. **34**:343, 1916.

13. Winkler, C., and Van Gilse van West, J.: Das Gehirn eines amaurotisch-idioten Mädchens, Verhandel. der Koninklijke Akademie van Wetenschappen te Amsterdam Deel XVII, No. 5, 1913.

14. Sachs, B.: J. Nerv. & Ment. Dis. **14**:541, 1887; Die amaurot. familiäre Idiotie, Deutsch. med. Wchnschr. **24**:33, 1898.

15. Schaffer, K.: Zur Pathogenese der Tay-Sachs' schen amaurot. Idiotie, Neurol. Cntralbl. **24**:386, 437, 1905; Ueber die Anat. u. Klinik der Tay-Sach'schen amaurot. familiären Idiotie mit Rücksicht auf verwandte Formen, Ztschr. f. d. Erforschung u. Behandlung. d. jügendlichen Schwachsinns **3**:19, 147, 1909.

16. Schaffer, K.: Neue Beiträge zur Mikro-morphologie u. anat. Charakterisierung der infantil-amaurot. Idiotie, Ztschr. f. d. ges. Neurol. u. Psychiat. **46**:1, 1919.

tion is rather a negligible factor or even absent altogether. Instead, he postulates an anomaly in the chemistry of the ganglion cell cytoplasm, an inhibition of myelinization, etc. Sachs [17] also changed his views, considering the condition a degeneration or abiotrophy. The majority (Kingdon,[18] Pointon, Parsons, Holmes,[19] Torrance [20] and others) look on amaurotic family idiocy as principally, if not exclusively, a degenerative disease process.

My studies of a case, type Tay-Sachs, seem to justify an opinion that amaurotic family idiocy is essentially a developmental anomaly in that the ganglion cells are defective in their growth and insufficiently supplied with the stainable (chromatophilic or Nissl) substance. In addition, they show more or less marked degenerative phenomena, both the developmental and the degenerative conditions being especially in evidence in the optic thalamus.

A study of the literature [21] shows that these two factors are also present in the other types—late infantile and juvenile—with the probable predominance, however, of the degenerative changes.

REPORT OF A CASE

History.—A boy, aged 13 months, of Russian Jewish parentage, was admitted, Dec. 3, 1917, to the Cook County Hospital, where he died, Jan. 20, 1918. The mother said that this was her first born child; she denied miscarriages, venereal and all familial nervous or mental diseases. The child was born after an easy and normal labor and was breast fed up to the age of 12 months; he was never "bright" and was "apparently blind," for he would turn the eyes only when spoken to or when a sound was made. He never sat up, could never hold the head up and was always content to lie quiet.

Examination.—The patient was a well nourished, robust boy with a square head (caput quadratum), prominent frontal and parietal bosses, open fontanels, prominent abdomen and marked atrophy of the muscles of the upper and lower extremities (except the biceps and adductors of the thighs). The tendon reflexes were present; the abdominal reflexes were sluggish; there was no Babinski sign. The pupils reacted to light; strabismus and nystagmus were absent. The outlines of the disks were clean cut; in the macular region was a grayish area with a bright, cherry red spot in the center. The blood vessels were normal. There was marked hyperakusis—slight sudden noises caused the child to throw

17. Sachs, B.: On Amaurotic Family Idiocy, J. Nerv. & Ment. Dis. **30**:1, 1903.

18. Kingdon, E. C., and Risien Russel: Infantile Cerebral Degeneration, Med. Chir. Trans. **80**:87, 1897.

19. Pointon, F.; Parsons, J., and Holmes, G.: A Contribution to the Study of Amaurotic Family Idiocy, Brain **29**:180, 1906.

20. Torrance, H.: Tay-Sachs' Disease: A Study of the Symptomatology and Pathology of Three Atypical Cases, Glasgow M. J. **97**:193, 263, 341, 1922.

21. Walter, F. K.: Ueber familiäre Idiotie, Ztschr. f. d. ges. Neurol. u. Psychiat. **40**:349, 1918.

the hands up. The spinal fluid was clear, under normal pressure and contained 5 cells per cubic millimeter. There was no globulin, and the Wassermann test was negative.

The child gradually lost weight, and died at the age of 14½ months.

Necropsy Findings.—The findings were: bed sores in the sacral region; absence of teeth; open anterior fontanel; normal thyroid and thymus glands; bilateral lobar pneumonia; dura adherent to the skull cap; the brain, hard, firm and heavy, retained its shape; it weighed 1,000 gm. The brain felt as

Fig. 1.—Long fibrils in the apical dendrite and the ventral horn cell body where they form a network overlapping the cytoplasmic reticulum shown in the cell to the right. Bielschowsky stain; X 560.

though it were hardened in formaldehyd. The lateral ventricles were not dilated and contained clear fluid. The spinal cord was "hard, firm and heavy."

Microscopic Examination.—Figure 1 shows some ganglion cell changes typical of this disease—the cell body swollen, the nucleus dislocated toward the practically normal apical dendrite; the rest of the dendrites either swollen or obliterated. Many cells therefore appeared round or oval.

The periphery of the cell body as well as the dendrites showed a mass of neurofibrils, but the center was represented by a delicate reticulum—a so-called cytoplasmic endocellular network. It was more dense around the nucleus, resembling the perinuclear ring described by Donaggio in dogs, rabbits and cats. In some cells, a mass of so-called long fibrils could be seen as if emanating from the perinuclear ring and crossing the cell body forming in some instances what Donaggio described as a perinuclear cap. The long fibrils in the dendrites, at the periphery and crossing the cell body, were stable formations, actual neurofibrils. The endocellular fibrils, in contrast, were very unstable, often appearing dustlike, as broken up, shapeless granules filling the larger portion of the cell body. Unlike the long fibrils, they stained well with almost any method—thionin, toluidin blue, silver nitrate, gold corrosive sublimate (Ramón y Cajal), etc. All these staining methods revealed a network of fine threads with formation of minute vacuoles, regular in shape, about equal in size and, as noted, somewhat condensed around the nucleus. The contents of the vacuoles or meshes, that is the interfibrillary substance, considered by Schaffer to be the hyaloplasm, did not stain with the foregoing methods, but were distinctly brought out with various hematoxylin stains (Weigert's and Ehrlich's, and especially well with Schaffer's modification of the Weigert-Pal staining method).[22]

Figure 2 shows the contents of the vacuoles stained with Schaffer's method. They appeared as minute granules, quite regular in shape, forming parallel rows and occupying the space of the cell body devoid of Nissl's substance. The latter was gathered (Fig. 3) in the upper portion, in the form of marked and well stained granules around a healthy nucleus rich in chromatin. The appearance of this portion of the cell body was that of a rather normal ganglion cell, in marked contrast to the rest which, packed with hematoxylinophilic granules, was decidedly abnormal. Some cells showed large amounts of other

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22. This method has been described by Schaffer (Arch. f. Psychiat. **64**:570, 1922) as follows: (1) Small pieces (from 0.5 to 1 cm. thick) are mordanted in a 5 per cent. solution of potassium bichromate in an incubator for a period of from two to three months or longer; (2) this is then embedded in celloidin or paraffin (from one to two weeks); (3) sections 6 microns thin, held on the slide with glycerin albumin, are again placed in a 5 per cent. potassium bichromate solution in an incubator for from three to seven days; (4) they are then washed thoroughly in water and (5) stained with Weigert-Kulschitzky's method for from twenty-four to forty-eight hours (in an oven); (6) then washed in water for several hours and (7) differentiated according to Pal's method; (8) put in a warm saturated solution of lithium bicarbonate until the stained areas become deep blue; (9) dehydrated and (10) counterstained by van Gieson's method; (11) washed slightly in water, and passed through alcohol and xylol, and embedded in Canada balsam. Equally good results may be obtained with a simpler method. Frozen sections passed through 80 per cent. alcohol are placed in Weigert's "Schnell-Beize" (fluorochrom 2 gm., potassium bichromate 5 gm., distilled water 100 c.c.) for twenty-four hours in an incubator; these are then passed through water and 70 per cent. alcohol into Weigert-Kulschitzky's hematoxylin for twenty-four hours, also in an incubator; washed in distilled water; differentiated according to Pal's method and passed through alcohol (95 per cent. absolute), carbolxylol and mounted in glycerin or Canada balsam. Sections stained in this manner may be counterstained with sudan III, scarlet-red, Alzheimer-Mann, and give instructive pictures.

granules that stained with sudan III, scarlet-red and osmic acid. However, the majority of the ganglion cells (cortex, spinal cord) appeared but slightly red when stained by some of the foregoing methods, while in some areas (corpus striatum and globus pallidus) the ganglion cells were packed with minute lipoid granules discerned only with a fairly high power lens (Fig. 4). Other areas, again, such as Ammon's horn and the occipital lobe, appeared much richer in such granules, while in the optic thalamus they were gathered in immense amounts, in the form of large drops. In short, some ganglion cells contained, besides the normal Nissl substance, vast numbers of other granules, some with strong affinity for hematoxylin, some for fat stains. The former

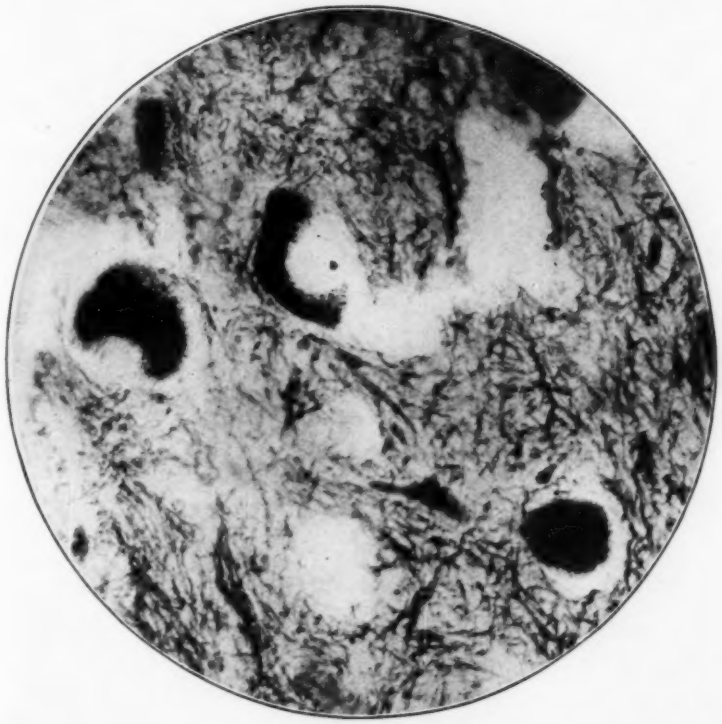

Fig. 2.—The ventral horn cells of the spinal cord. The larger, dark stained portion is filled with prelipoid (hematoxylinophylic) granules; the normal, perinuclear, portion is unstained. Schaffer's modification of Weigert-Kulschitzky's stain; X 200.

were classified by Alzheimer[23] as prelipoid, the latter as lipoid, substances. In some ganglion cells the Nissl substance, the prelipoids and lipoids were rather equally represented, but in some the Nissl bodies were either entirely absent or gathered in scanty amounts around the nucleus or at the periphery.

23. Alzheimer, A.: Ueber den Abbau des Nervengewebes, Neurol. Centralbl. **25**:472, 1906; Beiträge zur Kenntniss der pathol. Neuroglia und ihrer Beziehungen zu den Abbauvorgängen im Nervengewebe, Nissl-Alzheimer's Arb. **3**:401, 1910.

Such ganglion cells lost their characteristics—they were devoid of dendrites, the apical process was small, thin, or absent, the nucleus was dislocated, usually shrunken and devoid of chromatin. Especially numerous were such disfigured cells in the optic thalamus where they often appeared vacuolated with a nucleus (Fig. 5) always misplaced and often broken up. They appeared as mulberry bodies, and were described in amaurotic family idiocy by Globus as glia cells, but he does not specify the place of their occurrence. In many instances, they

Fig. 3.—Ventral horn cells of the spinal cord. The perinuclear area contains an abundance of normal Nissl substance, the rest of the cell body is unstained—the reverse of the condition shown in Figure 2. Toluidin blue; X 560.

much resembled gitter cells, from which it was not easy to differentiate them. In fact, many ganglion cells, on closer examination under very high power lens, showed as an aggregate of three or four minute gitter-cells (Fig. 6). Scattered in immense amounts among somewhat preserved but usually badly damaged ganglion cells, they gave to the field of vision a sieve-like appearance (Fig. 7), and in specimens stained with sudan III, scarlet-red revealed a vast accumulation

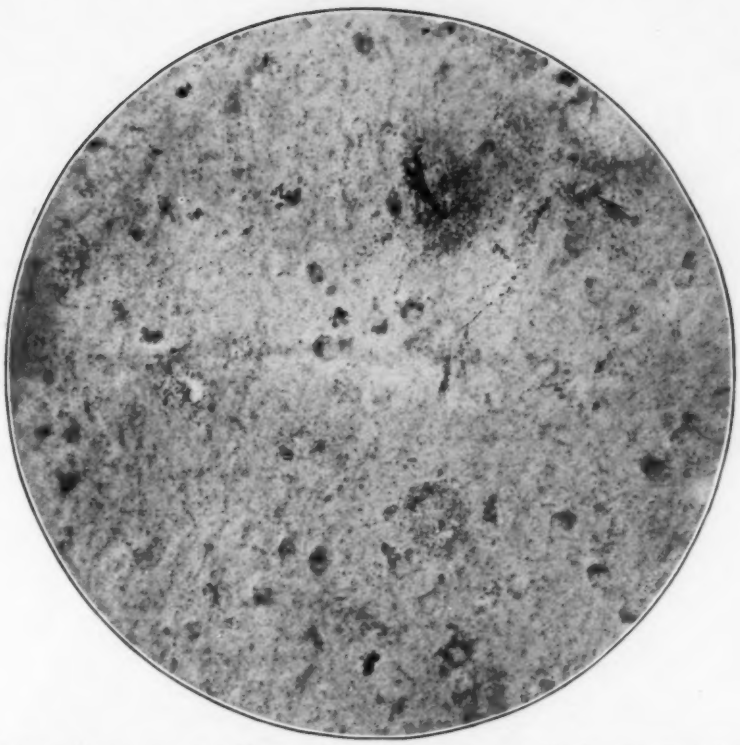

Fig. 4.—Putamen. Small droplets and granules of lipoids in the glia and ganglion cells. Scarlet red-hematoxylin stain; X 200. Compare with Figure 8.

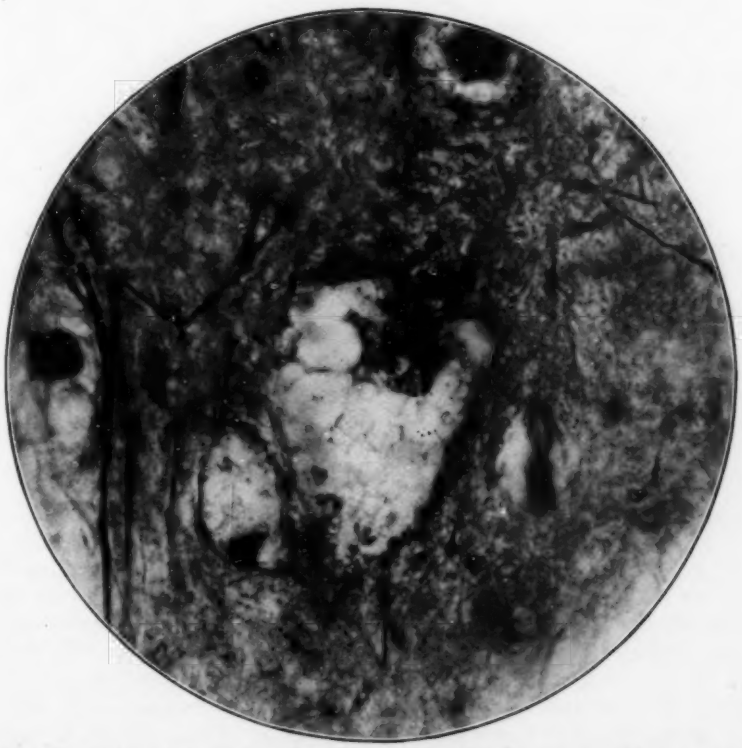

Fig. 5.—Optic thalamus. Ganglion cells with large vacuoles resembling mulberry-like cells. The central cell shows a nucleus rich in chromatin and dislocated to the periphery. The round cell adjacent to this cell below and to the left is a gitter cell. Nerve fibers are few. Bielschowsky stain; X 1200.

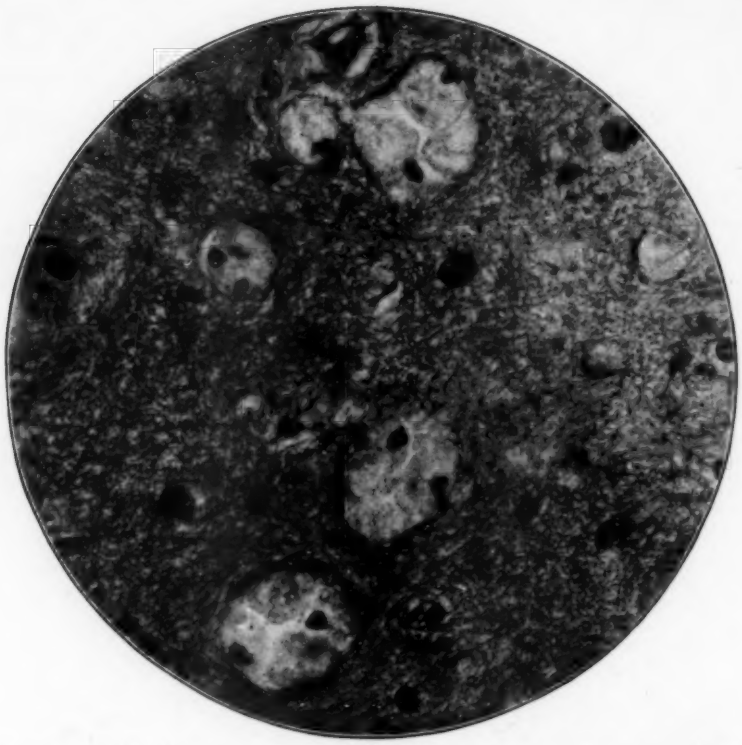

Fig. 6.—Optic thalamus. Conglomerates of gitter cells surrounded by a common capsule. The rest of the field is represented by fibrous glia tissue. Alzheimer-Mann stain; X 560.

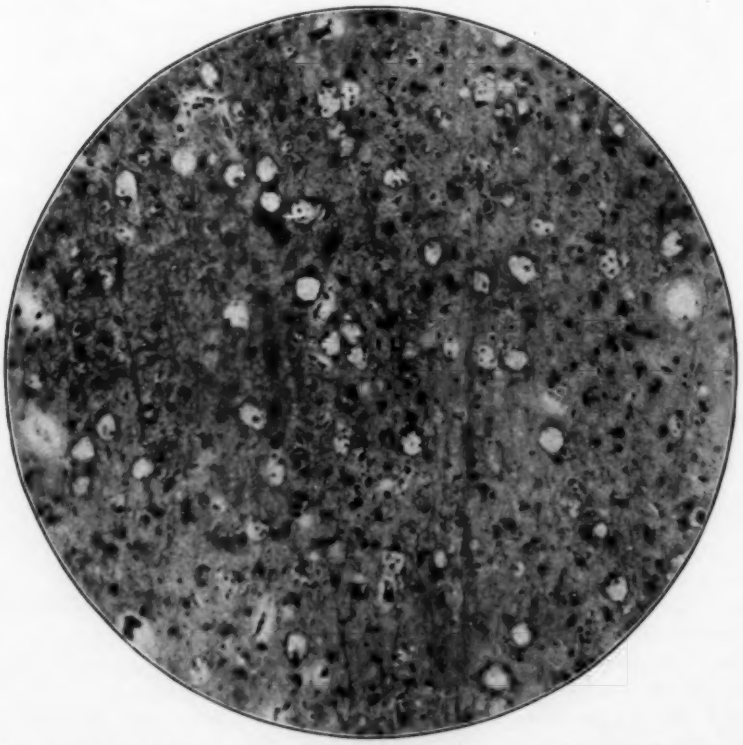

Fig. 7.—Sievelike appearance of the optic thalamus. The round pale bodies are gitter cells; the numerous black dots are cytoplasmic glia cells (the cytoplasm can be seen with a hand lens); nerve fibers, in the form of naked axons, are few; the rest of the field is taken up by fibrous sclerosed glia. Bielschowsky stain; X 100.

of fatlike substances covering the entire visual field (Fig. 8). Compared with the optic thalami, the rest of the cerebral areas appeared much less affected. Thus the cerebellum exhibited rather well preserved Purkinje cells, an excellent granular layer and a large amount of basket cells in the molecular layer. The latter, as well as the Purkinje cells, were disfigured, their dendrites expanded, thickened and swollen, but the nucleus, the so-called climbing ("Kletter"), mossy ("Moos") and other fibers were mostly well preserved. Like the rest of the ganglion cells, the Purkinje cells contained prelipoid substances, but no lipoids, and exhibited a rich supply of Nissl bodies.

The expanded cell bodies greatly crowded the visual field, occupying in some regions (Ammon's horn, spinal ganglia, Clarke's columns) all available

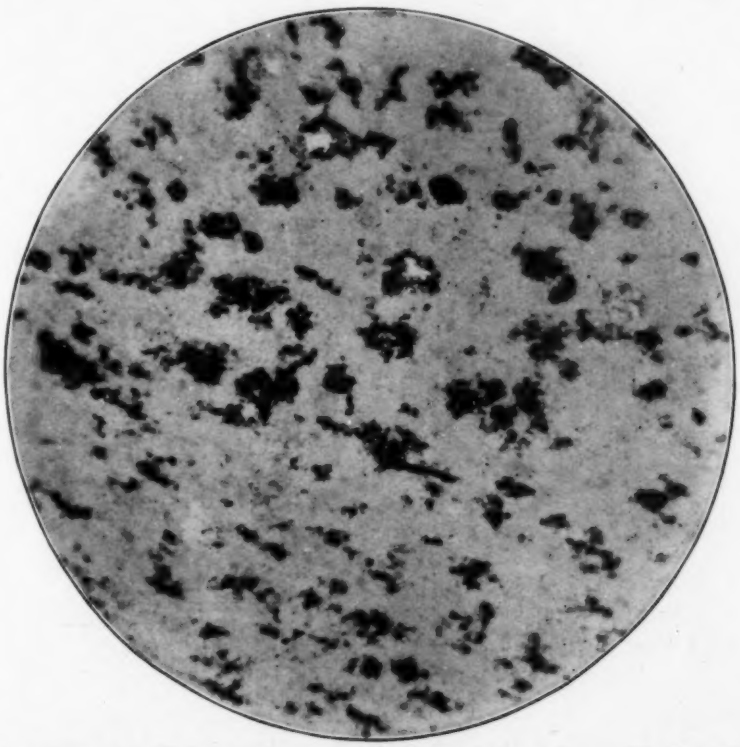

Fig. 8.—Optic thalamus. Masses of lipoids within the ganglion cell bodies. Scarlet-red hematoxylin stain; X 200. Compare with Figure 4.

space. They were even in the white substance, where they appeared as though pushed there from the overcrowded gray substance (developmental or heterotopic changes?).

The cell changes outlined were universal—in the motor as well as in the sensory portions of the central nervous system; in some regions more, in some less, pronounced. In the spinal cord, medulla and cerebellum, the changes were less advanced than in the cerebral cortex; in the latter, less than in Ammon's horn, reaching their height in the optic thalami.

A still more noteworthy feature was the varying intensity of the cellular lesions even in the same area. In the medulla, for instance, the cells of the

nucleus of the twelfth nerve were less involved than those of the olivary bodies, exhibiting a tolerably well preserved shape, good fibrillary structure and an appreciable amount of Nissl bodies, with excellent nuclei and quite visible, well developed processes. On the other hand, the cells of the inferior olivary bodies appeared markedly expanded, empty, almost devoid of stainable substance, a nucleus damaged, the cell bodies often resembling gitter cells.

Axons: Much as the cell bodies appeared changed, the axons and their prolongations, the nerve fibers with their myelin cover, were, as a rule, well preserved. Thus the nerve fibers exhibited good staining throughout the brain, although the myelinated fibers were by no means so dense or numerous as normally. In the optic thalamus, these were especially sparse, usually rep-

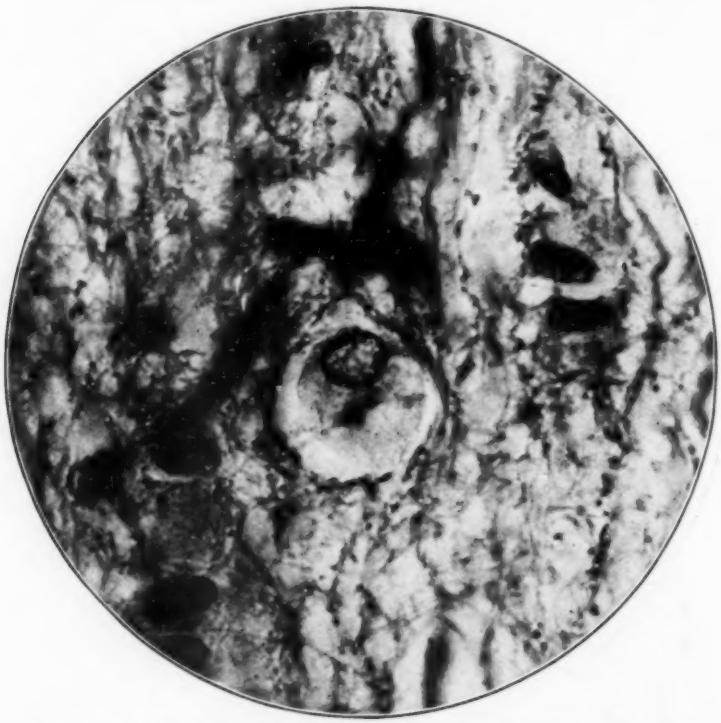

Fig. 9.—Longitudinal frozen section of the lateral pyramidal tract. The dark streaks are nerve fibers; the large vacuolated body with an eccentric nucleus is a myelophage; the other cells, with large nuclei, rich in chromatin are also gliogenous formations; the pale thin fibers are glia. Alzheimer-Mann stain; X 1200.

resented by a few naked axons. In the spinal cord, the rarefication of the nerve fibers was marked in the lateral columns, in the areas occupied by the pyramidal tracts, and to a lesser extent, in the anterior pyramids. Longitudinal sections of such areas stained by the methods of Alzheimer-Mann, Bielschowsky, or these two methods combined, exhibited a great mass of well preserved fibers, divided in many instances from each other by long stretches of glia tissue. This was usually in the form of delicate fibers (fibrous glia) harboring

numerous cytoplasmic glia cells. Occasionally, but rarely, there were also other signs of early nerve degeneration, such as formation of myelin globules, myelophages, etc. (Fig. 9).

Glia: In the cortex and in the pons where the ganglion cells were excessively crowded, the glia cells were rather inconspicuous. In fact, the first impression, especially on a superficial examination, was that there were hardly any glia cells when contrasted with the spinal cord, the molecular layer of the cerebellum or the medulla. In some areas—stratum zonale, the subependymal area of the medulla, and in general in the peripheral portions, there were as in the spinal cord, signs of glial proliferation (cytoplasmic glia, glitter cells

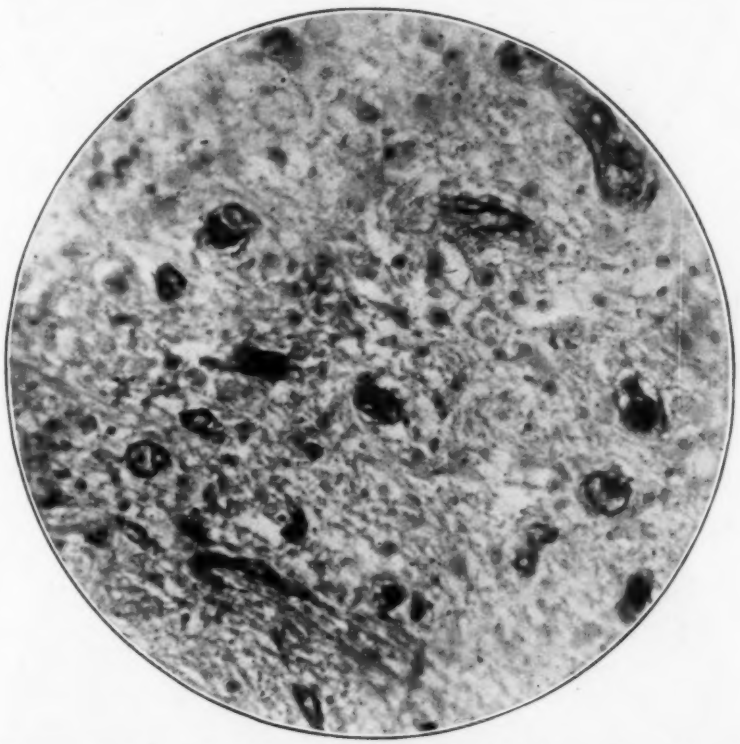

Fig. 10.—Proliferated glia tissue and blood vessels (dark stained formations) in the subependymal region of the medulla. Alzheimer-Mann; X 230.

and excessive formation of blood vessels) (Fig. 10). Some areas exhibited microscopic glial foci with total absence of nerve tissue, which was replaced by the cytoplasmic glia cells and reticulated fibrous glia (Fig. 11). In the spinal cord (sacral portion), the glia appeared as peculiar formations (Fig. 12), especially in the white substance, around the central canal and often around the capillaries. They showed under low power as round, coarsely granular bodies, scattered singly and stained well with Weigert's hematoxylin and the Alzheimer-Mann method, but showed no lipoids. As Figure 13 shows, these bodies are glia formations with an abundance of processes enveloping nerve fibers which probably are in the state of beginning degeneration.

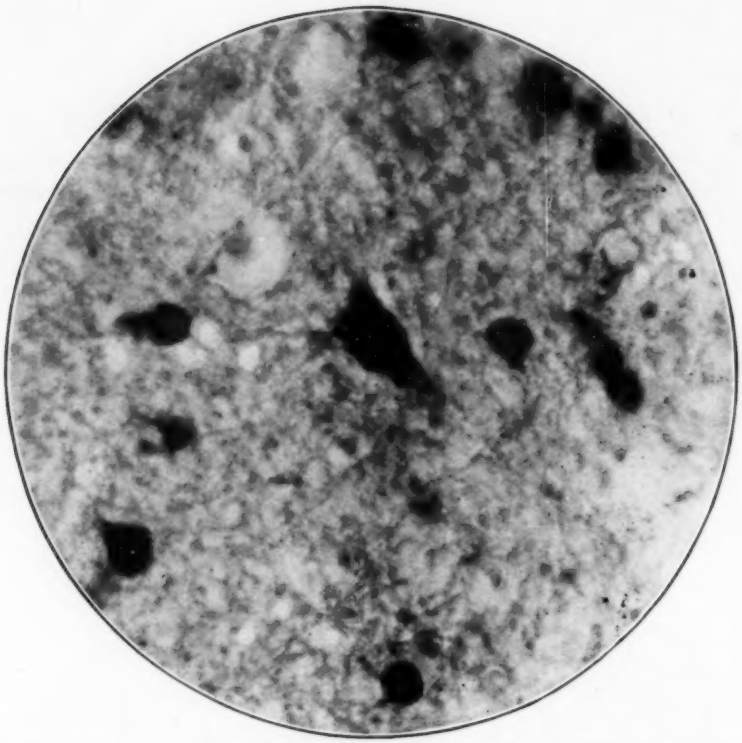

Fig. 11.—A microscopic focus of glia tissue with cytoplasmic large glia cells (dark stained cell bodies). Alzheimer-Mann; X 1200.

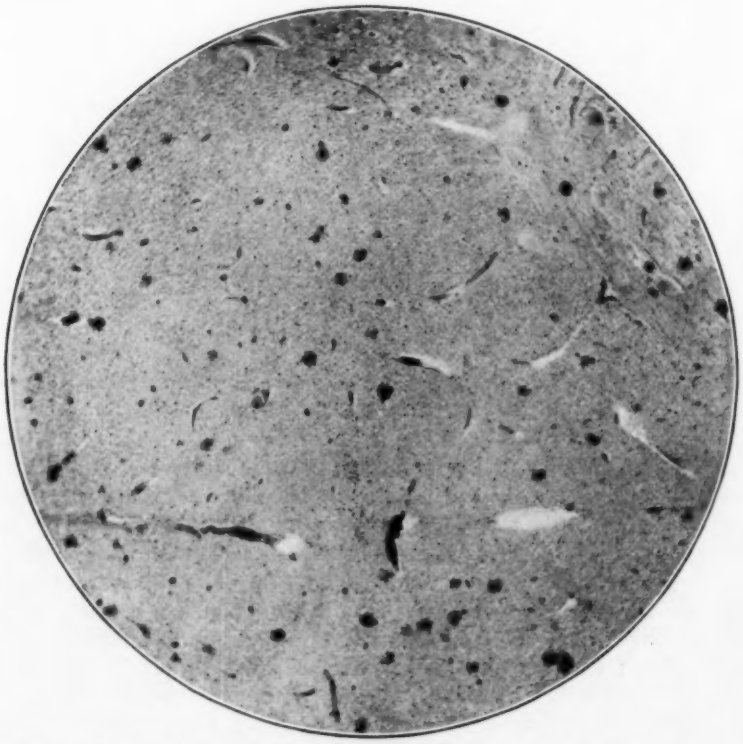

Fig. 12.—Small islands of glia tissue in the white substance of the spinal cord. Reproduced under higher magnification in Figure 13. Stain same as in Figure 11; X 60.

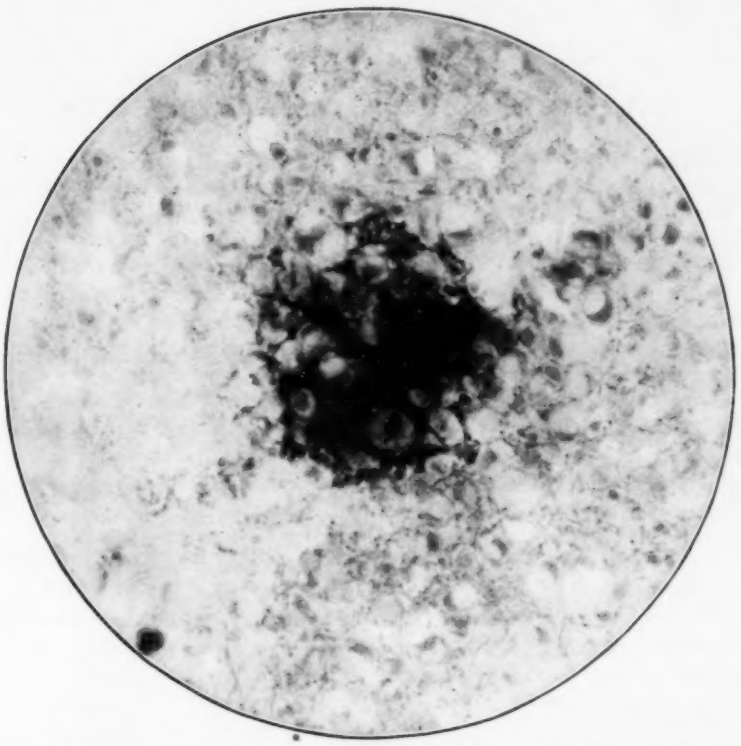

Fig. 13.—Same as Figure 12. Proliferated glia (dark thick fibers) enveloping single nerve fibers (paler structures with a dark axon in the center). Stain same as in Figure 11; X 1200.

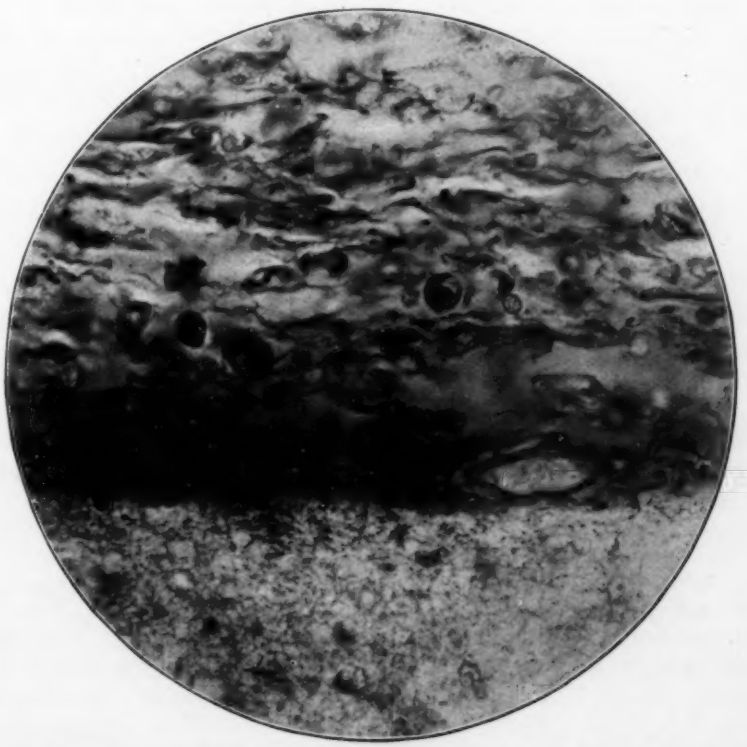

Fig. 14.—Hyperplastic pia with cellular contents explained in text. Stain same as in Figure 11; X 560.

Finally, there were areas (around the blood vessels, in Ammon's horn) in which the glia was rarefied and reticulated and harbored a large number of glia nuclei.

Mesodermal Tissues: Pia-Arachnoid, Blood Vessels and Choroid Plexus: The pia-arachnoid (Fig. 14) was hyperplastic, thickened and often homogeneous. Its somewhat distended meshes contained lymphocytes, macrophages filled with pigment, gitter cells packed with lipoids, fibroblasts—often of unusual size—homogeneous round amyloid-like bodies, an amorphous gray substance, numerous minute granules of pigment freely scattered over the connective tissue fibers and enormous proliferation of the mesothelial cells. Some lymphocytes were mere nuclei densely stained (pyknotic); some possessed an appreciable amount of cytoplasm, some contained two nuclei always rich in chromatin. The pial changes were present all over the brain and the spinal cord, but were especially marked around the cerebellum. The blood vessels of the pia were few, inconspicuous, not infiltrated and without other changes in their walls.

The tuft cells of the choroid plexus were increased in size, often vacuolated or granular, somewhat shapeless and contained a homogeneous nucleus, dislocated to the bottom. Some tuft cells appeared as gitter cells, while the stroma was, like the pia, covered with numerous pigment granules. The ependymal cells of the ventricles and the spinal canal were well developed, rich in chromatin, often hypertrophied and hyperplastic, resting on a broad homogeneous lining.

The blood vessels generally appeared normal, but in some areas were numerous. Their endothelial and adventitial cells were prominent and brightly stained. There were no proliferative phenomena, such as budding, nor inflammatory changes; the adventitial spaces, in many instances, were dilated and packed with lipoid substances.

General Summary.—There were: absence of inflammatory phenomena; diffuse ganglion cell changes, typical of amaurotic family idiocy, especially marked in the optic thalamus; rarefication of nerve fibers, with reactive glia phenomena of varying extent and intensity; accumulation of lipoid substances in the adventitial spaces of the blood vessels and the pia-arachnoid.

COMMENT

Of the outlined findings, the most striking occurred in the ganglion cells. Described by Sachs-van Gieson,[14] Hirsch,[24] Spiller[25] and more extensively studied by Schaffer,[26] they attracted much attention and caused a great deal of discussion. This is but natural, for the entire pathology of amaurotic family idiocy centers in the ganglion cells. Their changes are so conspicuous and widespread, those of the nerve fibers and the glia so strikingly in the background, that one unavoidably

24. Hirsch, W.: The Pathological Anatomy of a Fatal Disease of Infancy with Symmetrical Changes in the Region of the Yellow Spot (Warren Tay); Amaurotic Family Idiocy (Sachs); Infantile Cerebral Degeneration (Kingdon and Risien Russel), J. Nerv. & Ment. Dis. **25**:538, 1898.

25. Spiller, W.: A Pathological Study of Amaurotic Family Idiocy, Am. J. M. Sc. **129**:40, 1905.

26. Schaffer, K.: Tatsächliches u. Hypothetisches aus der Histopat. d. infantil-amaurot. Idiotie, Arch. f. Psychiat. **64**:570, 1922; also (footnotes 15 and 16).

gains an impression that the general, well established laws of neuropathology and even, as Sachs [17] thought, the neuron doctrine itself were at fault.

The balloon shaped, expanded cell bodies and dendrites show a distinctly fibrillary structure. They are made up of so-called long somewhat thick neurofibrils massed at the periphery, crossing the cell body and occasionally, as Figure 1 shows, forming anastomoses. They are analogous to the neurofibrils described by Bethe, Apáthy, Ramón y Cajal, Marinesco and others. The thin fibrils forming the so-called endocellular network are also considered by some (Donaggio) as being of nervous origin. This is denied by Economo [27] and Bielschowsky,[28] who look on this reticulum as an integral part of the cytoplasm, or as an artefact (Jäderholm [29]), caused either by a partial impregnation with silver or by adhesions ("Verklebungen"). The network, however, is not an artefact, nor is it made up of neurofibrils, for in my specimens there was no relationship between the thin threads of the reticulum and the actual, long, thicker neurofibrils. These were usually pushed to the periphery by the cellular contents, enclosed within the meshes which, formed by the threads of the cytoplasm itself, much resembled the reticular structure of so-called gitter cells. Just as in the latter a cytoplasmic network is formed to harbor lipoid substances, a similar reticulum is produced in the cytoplasm of a ganglion cell to harbor various abnormal, catabolic products, described as prelipoids—myelinoid, protagon or lecithinoid. The similarity between a classical gitter cell and a changed ganglion cell was sometimes so great that a differential diagnosis was not possible, especially when the contents of the ganglion cells were, as in the optic thalami, lipoids. In such instances, the ganglion cells were actually transformed into fat granule bodies, as Figures 6, 7 and 8 distinctly show.

In a great many cells, the cytoplasmic reticulum is broken up into a mass of small globules and granules somewhat resembling the condition known as chromatolysis, or broken up Nissl bodies. However, as Figure 2 shows, the granular masses are lodged only in the lower portion of the cell body, sparing the upper nuclear portion (Fig. 3) which harbors entirely normal Nissl bodies. Such an unequal arrangement of the coloring substance, being a common feature in amaurotic family idiocy, would be altogether inconceivable were the condition that of chromatolysis; for the latter could hardly affect only one portion of the cell,

27. Economo, C.: Zur normalen Anatomie der Ganglienzelle, Arch. f. Psychiat. **41**:158, 1906.

28. Bielschowsky, M.: Ueber die fibrilläre Struktur d. Ganglienzellen, J. f. Psychol. u. Neurol. **10**:274, 1908.

29. Jäderholm, G.: Endozelluläre Netze oder durchlaufende Fibrillen in den Ganglienzellen? Arch. f. Mikr. Anat. **67**:103, 1905-1906.

leaving the nuclear portion almost always intact. Bielschowsky and Schaffer (in his latest publications) do not consider the dustlike, granular appearance of the cell body as chromatolysis, but as a broken-up, cytoplasmic reticulum (considered by some as Donaggio's network). The absence of Nissl bodies is thought by Schaffer,[30] Sachs and Strauss [31] and Westphal [32] to be due to gradual absorption, by Bielschowsky to shrinking; by Mott [33] to progressive loss from the periphery to the center. Vogt [34] maintains that the morbid process, starting somewhere in the cell body, gradually invades its "interior," but some portions of the cell contain, he says, large amounts of Nissl bodies "embedded in an apparently normal cytoplasmic substance."

We are thus confronted in amaurotic family idiocy with a peculiar phenomenon—one portion of the ganglion cell is supplied with normal coloring substance gathered around a normal nucleus, the rest of the cell body contains abnormal products—prelipoids or lipoids. The former represents a nerve cell at the height of its development and growth, the latter a condition somewhat resembling a nerve cell in an embryonic state (before five months of fetal life). In the poverty of the dendrites, in the lack of Nissl bodies and in the presence of a cytoplasmic reticulum, it somewhat resembles a maldeveloped ganglion cell as described in human anencephalic monsters by Solovtzoff.[35] At any rate, the cell changes in amaurotic family idiocy are not acquired, for they are unlike any known pathologic or experimentally produced condition. Nor are they due to chromatolysis, to the disappearance, "shrinking," "absorption" or "using up" of Nissl bodies, because a larger portion of the cell body being in an embryonic or underdeveloped state, most likely never had them. They evidently attained their full development only around the nucleus, failing to develop in the rest. The characteristic feature of the ganglion cells in amaurotic family idiocy is therefore not their balloon, bottle, hour-glass or other distorted shape, but a deficiency in Nissl bodies, their replacement by abnormal chemical sub-

30. Schaffer, K.: Ueber Patho-physiologie eines neueren Falles (VIII) von Sachs' schen familiär amaurot. Idiotie, J. f. Psychol. u. Neurol. **10**:121, 1907. Also (Footnote 26).

31. Sachs, B., and Strauss, I.: The Cell Changes in Amaurotic Family Idiocy, J. Exper. Med. **12**:685, 1910.

32. Westphal, A.: Beitrag zur Lehre von der amaurot. Idiotie, Arch. f. Psychiat. **58**:248, 1917.

33. Mott, F., and Carlyll, H.: Seven Cases of Amaurotic Idiocy, Proc. Roy. Soc. Med. (Section of pathol.) **4**:3, 1911; Arch. of Neurol. (London) **3**:218, 1907.

34. Vogt, H.: Arch. f. Kinderheilk. **51**:1, 1909.

35. Solovtzoff, N.: Sur les difformitées congénitales du cerveau dans leurs rapports avec l'état des cellules nerveuses de la moelle, Nouv. Iconogr. de la Salpêtrière **11**:184, 1898.

stances and the presence of a well formed fibrillary structure. Such defectively formed cell bodies, however, are compatible with life, for they were found in adults. Thus Behr's [36] and Walter's [21] patients were aged 23 years; Rogalski's,[37] 26; Globus',[38] 21; and Sträussler's,[39] 36 years. The histologic differences found in the adult or the juvenile form—lack of marked swelling of the dendrites, a better preserved shape, a richer supply of Nissl bodies, accumulation of pigment and lipoids—are of rather minor importance and by no means striking. Whatever the differences, the fact remains that life is possible in the presence of ganglion cell changes such as are seen in amaurotic family idiocy. Life is rendered impossible when the ganglion cells, already defective developmentally, become degenerated, harboring vast amounts of lipoids. Schaffer [40] even suggested a classification of the cell pathology in various types of amaurotic family idiocy according to the pathologic contents of the ganglion cells. A prelipoid phase, with predominance of fuchsinophil and lecithinoid granules, is typical, he thinks, of the infantile group, the lipoid phase of the juvenile, the so-called osmic-reductive phase, of the adult. The more protracted the course, the more advanced and the riper, according to Schaffer, is the intracellular degenerative process.

In my case all these types were present, mainly in the optic thalamus. The majority of the cell changes corresponded with the first, prelipoid (called by Schaffer lecithinoid) stage; some in the occipital lobe and Ammon's horn exhibited features of the two other stages, but were rather scarce and were overshadowed by the elements of the first variety. In the optic thalami, the majority of the cell bodies showed features of the three stages of Schaffer combined. Here the cell destruction was the most extensive and intensive, not one cell body in some sections being preserved. The shape and size of the cells, as noted, were so changed that only comparative studies with many staining methods revealed their true nature. The optic thalami were the only structures in which the cell destruction was so far gone as to involve the entire parenchyma. This was represented (Figs. 6 and 7) by a fibrous glia covered sievelike by changed ganglion cells, scanty nerve fibers and naked axons.

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36. Behr, H.: Zur Histopath. der juvenilen Form der familiären amaurot. Idiotie, Monatschr. f. Psychiat. u. neurol. **28**:327, 1910.

37. Rogalski, T.: Zur Kasuistik der juvenilen Form der amaurot. Idiotie mit histopathol. Befund, Arch. f. Psychiat. **47**:1195, 1910.

38. Globus, J.: Ein Beitrag zur Histopath. d. amaurot. Idiotie, Ztschr. f. d. ges. Neurol. u. Psychiat. **85**:424, 1923.

39. Sträussler, E.: Ueber eigenartige Veränderungen der Ganglienzellen u. ihrer Fortsätze im Centralnervensystem eines Falles von kongenitaler Kleinhirnatrophie, Neurol. Centralbl. **25**:194, 1906.

40. Schaffer, K.: Arch. f. Psychiat. **64**:570, 1922.

Little is mentioned in the literature of the condition of the optic thalami. Bielschowsky and Tschugunoff [41] speak of their changes, and Globus found in the records of his third case (juvenile type) a note that there were remnants of optic thalami which were "gone." Microscopic studies could not be made as the brain was not preserved. It remains to be seen whether in every case of amaurotic family idiocy the optic thalami are as profoundly changed as in my case. I did find them markedly changed in specimens kindly sent me by Drs. Globus and Spiller. It remains also to determine whether the clinical phenomena—blindness, progressive deterioration, hyperakusis and certain motor phenomena—have anything to do with the pathology of this massive structure, the physiology of which is as yet so little understood. Study of the condition of the optic thalamus may explain the true nature of such cases as are reported by Spiller and McCarthy,[42] Spiller [43] and Sträussler.[44] In these cases, the clinical symptoms were not those of amaurotic family idiocy, although the ganglion cell changes were decidedly typical of this disease.

Assuming that the cell anomalies in the Tay-Sachs disease is a congenital defect compatible with life, we can understand the apparent absence of secondary degeneration and of glia changes. Both—secondary degeneration and reactive glia phenomena—would be as universal and as much in the foreground as the changes in the cells themselves, were these incapable of functioning. For a ganglion cell unable to maintain its function, because of pathologic changes, unavoidably leads to degeneration of the axon and subsequent proliferation of the glia. These, as noted, were present, but were comparatively mild and in the background. In other cases, such as those reported by Frey,[45] Spiller,[25] Naville,[46] Savini,[47] Globus,[38] some changes—axonal

41. Tschugunoff, S.: Zur Histopathologie d. infantil-amaurot. Idiotie, Arch. f. Psychiat. **69**:461, 1923.

42. Spiller, W., and McCarthy, D.: A Case of Internal Hemorrhagic Pachymeningitis in a Child of Nine Years, With Changes in the Nerve Cells, J. Nerv. & Ment. Dis. **26**:677, 1899.

43. Spiller, W.: A Case with the Symptoms of Cerebro-Spinal Meningitis with Intense and General Alteration of the Nerve Cell Bodies, but with Little Evidence of Inflammation, J. Nerv. & Ment. Dis. **28**:140, 1901.

44. Sträussler, E.: Zur Kenntniss der angeborenen Kleinhirnatrophie mit degenerativer Hirnstrangerkrankung des Rückenmarks, Ztschr. f. Heilkunde. **27**:30, 1906; also (Footnote 39).

45. Frey, E.: Path. Untersuchungen des Centralnervensystems in einem Falle von amaurot. famil. Idiotie, Neurol. Cntralbl. **20**:836, 1901.

46. Naville, F.: L'idiotie amaurotique familiale de Tay-Sachs, Schweiz. Arch. f. Neurol. u. Psychiat. **1**:314, 1917-1918.

47. Savini, C., and Savini, E.: Beitrag zur Aetiologie, Pathogenese u. path. Anat. d. Tay-Sach'schen familiären amaurot. Idiotie, Ztschr. f. Kinderheilk. **7**:321, 1913.

and lack of myelinization—were more widespread. In Savini's case, for instance, there was almost total absence of myelin in the occipital and temporal lobes, in the optic radiation, the reticular substance, the bundle of Vicq d' Azyr—the lack of myelinization having been combined with a diffuse degeneration of the ganglion cells. Globus found in his second case, that of a patient aged 25, a diffuse degenerative and sclerotic condition with diffuse myelin deficiency in the frontal and occipital lobes, optic thalami, Reil's island, corpus callosum, external geniculate bodies and the cerebellum. The changes were associated with pronounced glia proliferation—cytoplasmic and fibrous glia.

While some cases of deficient myelinization might have been due to aplasia, some were undoubtedly due to cellular destruction and degeneration as evidenced by the scattered reactive phenomena in the glia. However, such widespread myelin changes as were found in the foregoing cases are by no means common. In my case, they were rather scarce and altogether out of proportion to the number of ganglion cells changed but not destroyed. But they were in proportion to the number of ganglion cells actually destroyed, showing in rarefication of white substance, in destruction or disappearance of single nerve fibers replaced by microscopic glia foci and cytoplasmic glia cells (in the spinal cord Figs. 12 and 13). Where the cell destruction was most extensive and intensive, as in the optic thalami, the glia reaction was equally extensive showing as a glia scar (Fig. 7). On the other hand, in the cortex the glia was rather inactive, appearing as normal glia nuclei, without cytoplasmic cells or other signs of glia proliferation. This is in complete accord with the few ganglion cells and axons actually destroyed.

The glia, thus, is not diseased or degenerated, as assumed by Schaffer and Bielschowsky. The former [40] maintains that its lesion is as universal as that of the ganglion cells ("everything in the nature of nerve substance and neuroglia is at fault"); the latter, that the trophic function of the glia is at fault. On the contrary, it behaves as in any other pathologic condition, following in amaurotic family idiocy the general, well established laws of neuropathology.

The changes in the mesodermal elements—blood vessels and the pia-arachnoid—are like those in the glia, secondary. The lipoids resulting from the degenerated parenchyma are carried to the adventitial spaces of the blood vessels and thence to the pia-arachnoid, where they are found within gitter cells and where they provoke reactive phenomena —proliferation of fibroblasts, accumulation of mesothelial cells and lymphocytes—a condition discussed in some previous contributions.[48]

48. Hassin, G. B.: Studies in the Pathogenesis of Multiple Sclerosis, Arch. Neurol. & Psychiat. **7**:589 (May) 1922; Notes on the Nature and Origin of the Cerebro-Spinal Fluid, J. Nerv. & Ment. Dis. **59**:113, 1924.

As to the cause of anomalous condition of the ganglion cells one can but speculate. Schaffer [49] sees it in the deficiency of the hyaloplasm, resulting from a congenital weakness of the ectodermal layer; Mott, in the deficient formation of nucleo-proteids in the central nervous system; Bielschowsky,[5] in a pathologic ferment; Savini, Gordon [50] and Tschugunoff in endocrinal disturbances. None of these hypotheses can be verified by the neuropathologic findings. These only show that amaurotic family idiocy is a developmental anomaly associated with a degeneration especially of the optic thalami.

CONCLUSIONS

1. The main pathologic changes in amaurotic family idiocy are in the ganglion cells.

2. The changes are endogenous—first congenital and later degenerative.

3. The congenital changes are in the form of aplasia—a portion of the ganglion cell remains undeveloped, lacking in Nissl substance.

4. Some of the undeveloped aplasic ganglion cells ultimately become degenerated, causing secondary nerve degeneration and reactive glia phenomena.

5. Both the developmental and degenerative phenomena are especially marked in the optic thalami.

DISCUSSION

Dr. J. H. Globus, New York: Dr. Hassin's conclusions can hardly be considered as final, nor are they warranted by a study of a single case. I fully agree with him, however, that we are dealing here with a degenerative disease process engrafted on some form of structural malformation. In this connection, it is interesting to recall the early contributions of Sachs, in which he regarded this disease as an expression of some form of cellular maldevelopment, and, accordingly, termed it "aplasia corticalis cerebri," but a number of years later, in collaboration with Strauss, he was convinced of the degenerative nature of the pathologic process. While the degenerative features in the disease picture of amaurotic family idiocy are recognized by all who studied such material, the other factors, such as the aplasia of Sachs, the inherent malformation of Strauss, Hassin and others, are still open to investigation. Indeed, it is probable that certain components of the central nervous system in their early histogenesis have either failed to acquire certain properties or have developed certain defects which expose them to the injurious influence of some toxin, most probably of an endogenous character. Unfortunately, however, no definite proof has as yet been brought forward to support such an assumption.

The rather old conception that the pathologic changes in this disease are limited to the ganglion cells of the gray matter must be set aside in view of

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49. Schaffer, K.: Die allg. histopath. Charakterisierung d. Heredo-degeneration, Schweiz. Arch. f. Neurol. u. Psychiat. **7**:193, 1920.

50. Gordon, A.: New York M. J. **85**:294, 1907.

the recent contributions. I need only refer you to the investigations of Bielschowsky and to my own to remind you that dendrites and axis cylinders have also been found to show striking degenerative changes similar to those found in the cells in this disease, and that such changes may occur with the cell bodies fully intact. Schaffer also is inclined to the view that all the ectodermal derivatives of the central nervous system are involved, although there may be certain quantitative variations. It is difficult also to accept the assertion that the thalamus opticus is to be regarded as the seat of the most active destructive process, and that the course and the manifestations of this disease are determined by this unusual localizing feature. But here again recent investigations have clearly shown that no part of the central nervous system is immune to the disease, and it would appear that the more careful and complete our studies are, the less apt are we to stress the localizing character of the disease.

Dr. Bernard Sachs, New York: If Dr. Hassin will refer to some of my first articles on amaurotic family idiocy, he will find drawings almost similar to his slides. Even at that early date, van Gieson produced slides that helped me to recognize the tumefaction, the enormous increase in the cells and the almost entire absence of anything like a normal cell anywhere in the cortex.

At that time, I did not suspect that it was a process that affected practically every ganglion cell or every cell in the central nervous axis.

Some excellent histologic work has been done in connection with this subject. The work of Schaffer and Bielschowsky and that of Dr. Globus have brought the histologic studies to an almost perfect state.

I should like to correct the impression that the optic thalamus was particularly at fault in these cases, because I believe that is what happened in the brain Dr. Hassin examined, but in the other necropsies and examinations that have been made, the general consensus has been that there is really no particular section of the central nervous system that is more affected than the other. We have found extensive changes in the work done with Dr. Strauss that was reported in our paper in 1910. At that time, we confirmed the previous impression that it was not only the change in the basal ganglia and in the cortex, but that the changes were equally marked in the spinal cord.

I am very glad to have Dr. Hassin's corroboration of the interpretation that this is a degenerative process. Although I have maintained that view for thirty years, it would not hurt my feelings if someone brought forth evidence that there is some additional factor responsible for this early degeneration. I should be ready to receive evidence of that kind and, although the child born with this disease develops fairly normally until the age of 5 months and then collapses, there must be some additional factor besides the one of inheritance which contributes to the degeneration of the entire gray matter.

Dr. William G. Spiller, Philadelphia: I had the opportunity in 1904 of studying a case of amaurotic family idiocy, and reported my findings in the *American Journal of the Medical Sciences* for January, 1905. This study was made at a time when certain methods of staining the neurofibrils now common were not in use. I am able to confirm what Dr. Sachs has said, as every nerve cell that I examined was highly degenerated. In this paper, I called attention to the fact that these changes, so typical of amaurotic family idiocy, are not confined to this disease, and referred to my report in the *Journal of Nervous and Mental Diseases,* 1900, page 494, of two cases of other conditions in which almost similar, if not identical, changes were found throughout the central nervous system.

Dr. Hassin spoke of a nerve cell being pushed by the pathologic process in his case into the white matter of the spinal cord, and he regarded this displacement as a part of the pathologic process. I should be very loath to accept that as a part of the pathologic findings, because I have found heterotopia of the nerve cells in the white matter of the spinal cord as a maldevelopment of the spinal cord.

Dr. George B. Hassin, Chicago: I admit that many of the doubts and objections of Dr. Globus are well founded. To many of them an answer will be found in my paper; I shall only point out that the changes in the axons that he described are not primary lesions but secondary to a degeneration of the ganglion cells.

Dr. Sachs' statement that the condition is universal and does not show any preference for particular portions of the central nervous system, is important. The reverse was the purpose of my demonstration: namely, to show that in amaurotic family idiocy the optic thalamus is the principal structure involved. Not much attention has been paid to the thalamus in this disease, since the earliest workers — Sachs and van Gieson, Hirsch and Schaffer — laid stress on the fact that the characteristic pathologic feature is universal destruction of the ganglion cells. Then Bielschowsky said that the cerebellum is particularly involved, and now I claim that the optic thalamus changes are of primary importance. I am quite sure that such an opinion is fully justified and will be confirmed. Thus, Dr. Globus found in a protocol of a Hamburg case of this disease, a statement that the optic thalamus was gone, and in a case he kindly loaned me I found marked changes. Bielschowsky mentions the destruction of the ganglion cells in his late infantile case, and Tchungunoff of Moscow does the same in a case of Tay-Sachs type. If further studies confirm such findings, it would be interesting to contrast them with the pathologic changes in the corpus striatum, which I tried to do. The importance of such studies cannot be overestimated, for they may give us an idea of the function and clinical significance of the optic thalamus.

A STUDY OF EXTRADURAL SPINAL TUMORS*

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Extradural spinal tumors frequently present a certain vagueness in the symptoms not observed in intradural tumors, which may make early diagnosis difficult. This is due to mechanical factors involved in the interposition of the tough dura and the noncompressible cerebrospinal fluid covering between the tumor and the spinal cord. The interpretation of these mechanical factors may often make the differential diagnosis between extradural and intradural spinal tumors. This series of twenty extradural spinal tumors, six in the cervical and fourteen in the thoracic region, were studied from the point of view of determining the relationship between the mechanical factors and the symptomatology. The segmental distribution is indicated in Figure 1.

In order to bring together syndromes, presumably similar, a grouping of the tumors was made according both to the levels involved and to the pathology. They have, therefore, been grouped into cervical extradural tumors and thoracic extradural tumors, the latter being further subdivided into high, middle and low thoracic. The extradural tumors involving the cauda equina have been excluded, since the cauda equina growths, whether intradural or extradural, constitute a syndrome more nearly akin to peripheral nerve syndromes than those found in spinal cord tumors. Furthermore, secondary metastatic tumors of the vertebrae, which form such a relatively large group, have likewise been excluded from this study. These are usually readily recognized, and operation for their removal is seldom indicated.

From a pathologic standpoint, intradural extramedullary spinal tumors show a marked similarity, whereas the extradural group of spinal tumors are made up of a variety of neoplasms whose origin and morphology are widely separated and in whose type of growth there is considerable variation. It is obviously difficult to compare the clinical progress and symptoms of a chondroma with that of a sarcoma or angioma. The chondromas in this series were small discrete tumors, ventrally placed, and gave rise to a point of sharply circumscribed pressure on the cord. On the other hand, the extradural sarcomas extended in length over a number of segments and surrounded more than half to three quarters of the cord, causing both pressure atrophy of the bone with enlargement of the vertebral canal, and marked compression

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*From the surgical clinic of Dr. Charles A. Elsberg.

*Read at the Fiftieth Annual Meeting of the American Neurological Association, Philadelphia, June, 1924.

of the cord. Obviously, a tumor of such structure and extent would give rise to a clinical picture quite different from that produced by a circumscribed growth causing more limited pressure. It is consequently to be expected that in the syndrome of extradural spinal tumors a great variation in symptomatology and course would exist.

In this group of tumors, the pathology showed a wide variation, these conditions being found: sarcoma, osteosarcoma, fibroma, endothelioma, angioma, chondroma and osteochondroma. This may account for the rather dissimilar pictures which these extradural spinal tumors presented, and for the vagueness in extradural tumor symptomatology in general.

CERVICAL EXTRADURAL TUMORS

The six cervical extradural tumors were at practically the same levels, yet there was little similarity in either the presenting symptom or in the neurologic signs and the course of the disease. Table 1 shows the presenting symptom, the segmental level and the pathology of this group.

All were ventral tumors lying in front of the dentate ligament. In view of their ventral position, one would have expected to find, as an early symptom, weakness and atrophy due to pressure on the ventral gray column. In two of the six cases, weakness of the lower extremity on the side of the tumor was the first complaint, and only later was weakness and atrophy noted in the distribution of the segments involved. In three, first numbness and then weakness referable to the distribution of the segments or their roots was found. In one, the third case in Table 1, weakness and atrophy of the interossei and forearm muscles

TABLE 1.—*Presenting Symptom of Cervical Group of Extradural Spinal Tumors*

Segmental Level	Tumor Type	Presenting Symptom
Cervical VII–VIII	Melanosarcoma	Numbness and tingling of fingers
Cervical VII–VIII	Fibrosarcoma	Weakness of right leg
Cervical III–VII	Fibroma	Numbness and weakness of right hand
Cervical VII	Chondroma	Weakness of left leg
Cervical VI	Chondroma	Pain in both legs and back of chest
Cervical VII	Chondroma	Numbness in tip of index finger followed by pain in forearm along preaxial border; pain described as if he had held a cake of ice in his hand

were present, although the tumor at operation was found to be well above the segments supplying these muscles. The tumor in this case extended obliquely across the cord from the third to the sixth cervical segment, whereas the atrophy was specially in the distribution of the eighth cervical and first thoracic segments. As evidence that these particular segments were not compromised by the tumor, Horner's cervical sympathetic syndrome was not present. In one patient, the seventh and eighth cervical roots were involved in the tumor and in another the

seventh nerve root formed a sharp angle as it passed over the tumor. This relationship of the nerve roots to the tumor might account for the numbness and pain of which these patients complained. It was fortunate that in this series of cervical tumors there were two chondromas

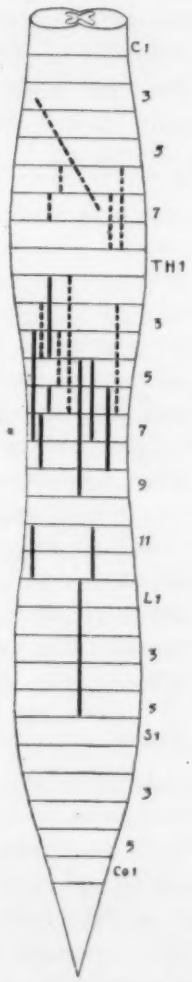

Fig. 1.—Segmental distribution of extradural spinal tumors. Broken lines indicate those tumors which were ventral to the dentate ligament.

almost similar in size, segmental level and position. In spite of the ventral position of both of these tumors and the pressure on the sixth and seventh cervical segments, the outstanding symptom was weakness in the lower extremities and difficulty in walking. From the position of both, pressure should have been exerted on the ventral gray column before the pyramidal tract could have been implicated. It is perhaps

possible that pressure gradually applied to the ventral gray column causes less disturbance in the function of the gray than when exerted on the long and more vulnerable fiber tracts, the latter being more susceptible to mechanical effects than the nuclear masses. In both of these cases, early evidence of disturbance in the pyramidal and spinothalamic tracts was found, viz.: weakness and stiffness in the lower extremities, together with a burning sensation, referable to the lower extremity and trunk of the opposite side in one case, and to the inner aspects of the opposite arm in the other.

TIME INTERVAL BETWEEN PRESENTING SYMPTOM AND OPERATION

The duration of the disease is somewhat closely associated with the size and consistency of the tumor. In the two hard unyielding chondromas compressing the cord rather sharply, this time interval was rather short. Before operation this might have led one to the belief that the rapid appearance and progress of the signs was due to a rapid malignant growth rather than to a relatively slow growing, benign tumor. It is possible that such hard tumors may give rise to no signs until they have compromised the entire free space of the vertebral canal, and then, when the cord is once compressed, due to the unyielding advance, the sequence of events is rapid and progressive. Both of these chondromas were relatively small as compared with the other cervical tumors. In the latter, the time interval between the presenting symptom and operation was approximately five times as long. The time interval for the chondromas was three and a half months and for the remaining cervical tumors eighteen months.

CONDITIONS WHICH MODIFY THE PRESENTING SYMPTOM

Frequently, spinal cord tumors of the cervical region show some spasm and rigidity of the neck muscles with limitation in movement. This symptom was not found in these cervical tumors. In two of the patients, movement of the neck or activity in general increased the pain, a symptom which is not rare in extradural tumors irrespective of the level. In one patient, lying down increased the pain rather than relieved it. It is possible that in a ventrally placed tumor pressure is exerted on the spinal cord when a recumbent position is assumed, when the cord tends to fall backward and on the tumor, while when the patient is standing, such pressure is not exerted. Unfortunately, the effects of such movements as sneezing and blowing the nose, etc., have not been recorded in all the histories. These sudden movements are associated with a marked rise of intrathoracic intra-abdominal and intracranial pressure, which causes a marked rise in cerebrospinal fluid pressure. When an obstructing mass, such as a tumor, blocks the sudden downward

movement of the cerebrospinal fluid, exquisite paroxysms of pain may be produced. The effect of sneezing and blowing the nose is such an important point in the history that it is worthy of record.

SENSORY CHANGES

In two of the cervical tumors, the spinal roots were involved, and yet in neither of them did anesthesia or hyperesthesia referable to the roots implicated exist; on the other hand, in a third patient, marked hyperesthesia in the fourth cervical dermatome existed, although at operation no involvement of the fourth cervical or any other nerve root was found.

The sensory changes in one of the chondroma cases was of great interest. This patient showed a loss of pain and temperature only. Here a small pointed chondroma was so situated as to compress a sharply circumscribed area of the cord in the region of the left spinothalamic tract, which gave rise to these sensory findings.

About the first symptom in this patient's history was a burning sensation over the right tibial crest, which gradually ascended, involving the leg, thigh, and half of the trunk up to the level of the nipple. This burning sensation was worse when walking and was confined entirely to the right side. The fact that this dysesthesia was referred to a definite region of the extremity and was followed by a gradual progression in more or less dermatomic sequence would tend to support the view offered by Dr. Elsberg [1] that the spinothalamic tract, like the dorsal tracts of the cord, is laminated; the fibers for any definite dermatome have a definite anatomic position within the tract. On account of the crossing of these fibers in the cord, those for the more distal dermatomic area lie toward the periphery, while the higher segmental levels are added on the medial side.

THORACIC TUMORS

In this group of extradural tumors, four originated primarily in the vertebrae and will be considered as a separate group. Of the remaining tumors, five were in the midthoracic region, involving approximately segments five, six, seven and eight, two were high thoracic tumors, and two were in the low thoracic region.

Midthoracic Extradural Tumors.—This group of tumors were placed anatomically, all being dorsal or dorsolateral and involving approximately the same levels; yet in spite of this, there was little correspondence in the course or progress of the symptoms. However, the presenting symptom in all, with one exception, was very much alike,

1. Proc. Forty-Seventh Annual Meeting Neurol. Assn., Atlantic City, 1921.

viz., pain in the back referred around the chest and variously described as "sharp, like a knife," "agonizing" and "severe" (Table 2). In one instance, weakness in the legs was the presenting symptom, pain being absent throughout the entire course of the disease. In this patient, the tumor was dorsally placed, yet no pain was present. Pain may be present or absent in ventral tumors as in dorsal tumors. Dysesthesia other than burning and cold sensations was encountered, and the latter, frequently noted in ventral tumors, were not present in a single instance of the dorsal tumors.

Bladder and rectal disturbances were present in all but, as previously pointed out,[2] did not appear until extremely late, usually shortly before the patient was operated on. In each the degree of cord compression was severe; all were completely paralyzed and bedridden, but bladder and

TABLE 2.—*Presenting Symptom of Midthoracic Group*

Segmental Level	Tumor Type	Presenting Symptom
Thoracic V-VIII	Sarcoma	Pain in back below shoulder blade; sharp and kife-like; unable to lie down or sit down
Thoracic V-VII	Angioma	Agonizing backache, worse on movement and menstrual period
Thoracic VII-VIII	Fibrosarcoma	Pain in left side of chest behind and beneath angle of scapula
Thoracic V-VI	Tuberculosis	Radiating and shooting pains in the back and extending on both sides to the front
Thoracic VI-VIII	Sarcoma	Weakness in legs, limping; no pain

rectal signs did not occur until nearly the end of the disease. In the cervical group, bladder and rectal signs occurred in only one of the cases, and then only when the cord compression had become extremely severe. There seems to be no doubt that bladder and rectal disturbances are not encountered until marked cord compression takes place, sufficient to disturb the short neuron visceral pathway with its numerous relays which, as Davis [3] pointed out, ascends as a juxtagriseal pathway. Because of the numerous relays and short neurons, this pathway is usually not interrupted until compression of the cord is extreme.

No common syndrome was made out in this midthoracic group in spite of their common level. Frequently in the postoperative course, in cases in which there were tumors in the region of the fifth thoracic and seventh thoracic segments, there was marked abdominal distress and distention, but no reference was found in the histories to any visceral disturbance which we felt could be considered as a characteristic symptom

2. Stookey, Byron: A Study of Bladder and Rectal Disturbances in Spinal Cord Tumor, Arch. Neurol. & Psychiat. **10**:519 (Nov.) 1923.

3. Davis, L. : Am. J. Physiol. **59**:321, 1922.

of this group. It is possible, however, that our histories were incomplete from this standpoint, and sufficient enquiry referable to the visceral symptomatology had not been made.

In one case, a tuberculous cyst presented in the vertebral canal caused cord compression similar to that of an extradural neoplasm. The signs and symptoms referable to the cord were identical with the tumor cases of this group, with the exception that local pain in the region of the diseased vertebrae was more marked than is usually found in extradural tumors.

In one angioma of the low thoracic region, there were points of particular interest. This patient gave a definite history of marked increase in both symptoms and signs during each menstrual period. The complaints were looked on with some scepticism until after the operation, when it was seen that the tumor had large vascular spaces which probably shared in the general hyperemia of the pelvic structures during the menstrual period. Thus the size of the tumor fluctuated, materially increasing at the menstrual period, to give rise to a corresponding increase in cord compression and cord signs.

High Thoracic Tumors.—In this group there are two tumors, one involving the first, second, third and fourth segments, and the other the second and third segments, one an endothelioma and the other an angioma, one ventral and lateral, and the other dorsal and lateral, both approximately of the same size. In the patient whose tumor was ventral, the presenting symptom was burning pain in the toes. Two and a half years after the onset the fourth and fifth toes were amputated, presumably for the relief of this symptom. The pain nevertheless continued, and nearly seven years after onset she began to have severe pain in the abdomen, radiating down both legs. Gradually her limbs became weak. Here again a ventral tumor pressing on the spinothalamic tract gave rise to a burning sensation. In the second patient with an angioma dorsal to the cord, pain referable to the upper part of the chest and legs was the presenting symptom. This continued at intervals for a year.

Neither of these tumors involved the nerve roots, yet a definite band of hyperesthesia and hyperalgesia was found, which, however, did not correspond to the level of the tumor but to one, two or three dermatomes below the level of the tumor. In tumors not involving nerve roots, hyperesthesia and hyperalgesia in a narrow dermatomic area immediately above the level of the tumor is, of course, relatively common, but hyperesthesia and hyperalgesia some distance below the level of the tumor is uncommon.

Low Thoracic Tumors.—At this level, two tumors of approximately the same size, similarly placed and at identical levels, gave rise to pre-

senting symptoms quite dissimilar. In one, the presenting symptom was a constant pain in the back, and in the other, a staggering and drunken gait, both gradually progressive without intermission. No particular symptom was found that was common to both patients or indicative of involvement of these segments. A level was determined only when sensory changes were finally present. Movement made the pain worse in one instance and had no effect in the other. Toward the end of the disease, dysuria occurred in one end and a sense of tenesmus in the other.

TABLE 3.—*Presenting Symptom in Primary Vertebral Tumors*

Segmental Level	Tumor Type	Presenting Symptom
Thoracic IV-V	Sarcoma	Pain in both legs lasting three months, followed by numbness and tingling beginning in right foot and extending upward
Thoracic VI	Sarcoma	Severe pain between shoulders and around the chest below the nipples
Thoracic I-IV	Sarcoma	Pain in chest and upper part of abdomen
Thoracic X-XII	Osteochondroma	Pain in lower lumbar region and referred down the leg

PRIMARY TUMORS OF THE THORACIC VERTEBRAE

The duration of the disease in this group was extremely short; it averaged about eight months, but was really less since the diagnosis of cord compression was made in one, ten months before operation was performed. The operation in this instance was delayed because the mediastinal growth was at first considered malignant, and laminectomy was delayed until it was believed that the growth was not malignant. If this period be deducted, the time duration of the disease in this group is less than five months, while that for the extradural tumor group as a whole is approximately twice as long.

TABLE 4.—*Time Duration from Presenting Symptom to Operation*

Extradural Tumors (Primary vertebral tumors excluded)	Average in Months
Sarcoma	7
Osteosarcoma	7.5
Angioma	21
Fibroma	20
Primary Vertebral Tumors	
Chondroma	3.5
Osteochondroma	4
Sarcoma	7
Total average	9

The time interval from the presenting symptom to operation in the extradural group is considerably shorter than that found in intradural tumors. The average duration of each pathologic group of extradural tumors is given in Table 4. It will be seen that the total average duration was approximately nine months. This is a shorter period than is

generally found in intradural growths. Rapidity of the symptom increase in a suspected spinal cord tumor should, therefore, make one consider the possibility of extradural rather than intradural tumor.

In two of the cases, rapid interference in cord conduction took place with relative suddenness; in one, complete paralysis followed in less than five days; and in the other, in less than ten days. Both of these patients were able to get about to within a few days before becoming bedridden. In connection with this sudden change in the symptom, sharp burning pain occurred and was referred from the back through the chest. In both, the tumor was found at operation to extend into the chest. The accession of this symptom of through and through pain of a sharp, boring character may be referable to the sudden breaking through of the tumor into the thoracic cavity. In neither of these cases was the sudden shutting off of cord conduction due to pressure on the cord, for at operation the cord seemed to be compressed but little. The tumor in both was extremely soft and vascular, and in one the pressure exerted on the dura appeared to be negligible. The usual explanation given for the sudden blocking of the cord conduction has been interference with the circulation of the cord. We have no proof that this is the explanation, but we do know that the amount of compression of the cord could not have given rise to the symptoms which indicated severe interruption of cord conduction.

Such sudden changes in the course of the disease are interesting. They may be due to causes other than interference with the vascular supply of the cord. A tumor arising in the bone and protruding into the vertebral canal may cause at first moderate cord compression, then breaking through the bony shell may give rise to a sudden accession of signs. Or a tumor may be held behind a nerve root or a dentate ligament, and in breaking free from restraint of either of the structures cause marked compression of the cord. Finally, the fluid covering of the cord may serve as a barrier until eventually it is completely shut off, and compression is then exerted directly on the cord without the interpositon of the fluid buffer.

It is interesting to note that a local malignant growth of the bone may give rise to the early local signs. Even local tenderness to deep pressure may be entirely absent. Although the presenting symptom may be due to pressure on the cord and referred to distal body segments, at some period during the course of the disease signs referable to the local process become manifest.

The roentgen-ray changes in this group of tumors were noteworthy. In two, maximum changes were noted in the mediastinum and not in the vertebrae concerned. Changes in the bony structure of the vertebrae were present in the remaining two cases, but it was doubted by the

roentgenologist that these changes were of pathologic significance. It is possible that if these plates had been studied by those familiar with the physical findings, the structural changes might have been considered of greater significance. In one, the roentgenogram showed the sixth thoracic vertebra to be smaller in all dimensions, although no absorption or destruction of the bone was noted. At operation, this proved to be a sarcoma which not only involved the sixth thoracic vertebra but probably extended into the mediastinum, although the latter fact was not determined with certainty. In another case, a palpable mass adjacent to the tenth, eleventh and twelfth thoracic vertebrae was diagnosed by roentgen ray as an osteochondroma, the diagnosis being substantiated at operation.

Of the primary bony vertebral tumors, three were chondromas and naturally showed nothing in roentgenograms. One osteochondroma was visible, and of the sarcomas, three showed significant changes which, while not pathognomonic of sarcoma, were at least sufficient to cause suspicion for further consideration.

ROENTGEN-RAY CHANGES IN EXTRADURAL SPINAL TUMORS OTHER THAN PRIMARY VERTEBRAL TUMORS

These changes were secondary; there was either an enlargement of the vertebral canal with the formation of what appeared to be a cyst or a slight scoliosis in the region of the tumor. In a fibrosarcoma in the cervical region, destruction of the lower cervical vertebrae was noted which led the roentgenologist to suspect the possibility of a bone cyst. A smooth cavity, relatively large, without erosion, formed presumably by pressure of the tumor, was found at operation. Since this patient was only 11 years old, this pressure atrophy may have been due to the fact that her vertebrae were not dense, and consequently atrophy could take place more readily.

In almost all of the extradural sarcomas, marked enlargement of the vertebral canal was noted at operation, the vertebral arches in some instances being reduced to a mere shell. This enlargement of the vertebral canal has not been sufficiently appreciated in the roentgen-ray examinations. Perhaps the technical difficulties in taking lateral roentgenograms of the thoracic vertebrae may account for failure to bring out this enlargement. Enlargement of the canal should, however, be looked for, and, when found, its significance recognized.

In three of the large extradural sarcomas of the midthoracic region, a slight scoliosis in the region of the tumor was noted but considered of no significance. However, since the scoliosis in both instances was definitely in the region of the tumor, it is perhaps plausible to consider the scoliosis as being due to the presence of the tumor.

TENDERNESS OF THE SPINE AS A DIAGNOSTIC SIGN

In a previous communication, Dr. Elsberg and I called attention to the value of tenderness of the vertebral spines as a diagnostic point in the differential diagnosis of spinal cord tumors and malignant disease of the bone, particularly metastatic disease. In this group of extradural tumors, tenderness of the vertebral spines was found in only one patient with primary tumor of the vertebrae. In this patient, tenderness of the sixth and seventh thoracic spines was noted, whereas the tumor was beneath the third, fourth and fifth and part of the sixth. It would seem that if tenderness of the spines were due to bone disease, the fourth and fifth vertebrae, which were the more involved, should have shown the more tenderness. The tender vertebrae corresponded instead to the compressed segments of the spinal cord. It may be that in this instance the tenderness of the spine was due to involvement of the spinal segments which supply the vertebrae, and in this respect conforms to the type of tenderness found in intradural diseases. In the remaining six patients with primary tumors of the vertebrae, tenderness was not elicited. Tenderness as a diagnostic sign in this group of vertebral tumors is consequently of little value (Table 5).

TABLE 5.—*Value of Tenderness of the Vertebral Spines as a Diagnostic Sign; Extradural Spinal Tumors—Primary Vertebral Tumors*

Tumor	Segmental Level	Spines and Arches Removed	Spines Tender
Sarcoma	Thoracic VI	.....	Not stated
Chondroma	Cervical VI, VII	.....	No tenderness
Chondroma	Cervical VI	.....	No tenderness
Chondroma	Cervical VII	.....	No tenderness
Osteochondroma	Thoracic X, XII	.....	No tenderness
Sarcoma	Thoracic V, VI, VII	Thoracic III, IV, V and part of VI	Thoracic VI and VII
Sarcoma	.....	.....	Not stated

Summary: Total number of patients, 7; number of patients with tender spines, 1; number of patients not stated, 2; number of patients without tender spines, 4.

In extradural spinal tumors other than those primarily of the vertebrae, tenderness was found in six cases. In three, the tender spine corresponded to both the segments and the vertebrae; in two, to the distribution of the segment, and in one only to the vertebrae beneath which the tumor was situated (Table 6). In one, the tenderness was five spines lower than the level of the tumor, and in another, restricted to the vertebra beneath which the tumor lay although the vertebra in this patient was not involved.

In the cervical and upper thoracic regions of the cord, the vertebrae and segments approximately correspond, and consequently, even if tender spines were present in these regions, they would not be of help in the differential diagnosis between malignant disease and extradural or intradural spinal tumor.

TABLE 6.—*Value of Tenderness of the Vertebral Spines as a Diagnostic Sign; Extradural Spinal Tumors Other than Primary Vertebral Tumors*

Tumor	Segmental Level	Spines and Arches Removed	Spines Tender
Sarcoma.....	Thoracic V, VI, VII, VIII, IX	Thoracic IV, V, VI, VII	Thoracic V, VI, VII
Sarcoma.....	Thoracic VI, VII, VIII	.....	No tenderness
Angioma.....	Thoracic X, XI	Thoracic VI, VII, VIII	Thoracic X, XI
Fibroma.....	Thoracic X, XII	.....	Not stated
Fibrosarcoma.	Thoracic XI, XII	.....	Not stated
Fibrosarcoma.	Thoracic VI, VII	Thoracic IV, V, VI	Thoracic V
Angioma.....	Thoracic V, VI	Thoracic II, III, IV, V	Thoracic VI
Endothelioma.	Thoracic IV, V	Thoracic II, III	Thoracic VIII
Fibrosarcoma.	Cervical VI, VII, VIII	Cervical V, VI, VII, Thoracic I	Cervical VI
Sarcoma.....	Thoracic VI, VII, VIII	.....	No spines tender
Fibroma.....	Cervical III, IV, V, VI, VII	.....	No tenderness of the cervical spines; movements free in all directions
Melanosarcoma	Cervical VI, VII	.....	No tenderness of the cervical or upper dorsal spines

CEREBROSPINAL FLUID FINDINGS

Xanthochromia was not found in this group of cases unless marked pressure of the cord was present, and never without an increase in the globulin content. On the other hand, a marked increase in globulin was found in all but one instance of marked compression irrespective of xanthochromia. Yellow fluid was not encountered in any block above the fifth thoracic segment, even though the pressure was sufficient so to compress the cord that it was little more than a thin band. In view

TABLE 7.—*Cerebrospinal Fluid Findings in Primary Tumors of the Vertebrae*

	Slight Pressure			Moderate Pressure			Marked Pressure			
	Cells	Globulin	Color	Cells	Globulin	Color	Cells	Globulin	Color	Level
Sarcoma.....	3	±	—	...	..	..	...	...	..	Thoracic 6
Sarcoma (giant cell).....	...	..	..	...	..	..	0	3+	Clear	Thoracic 1-4
Osteochondroma..	...	..	..	...	..	..	...	...	..	Thoracic 9-12
Sarcoma.....	...	..	..	...	..	..	0	4+	Clear	Thoracic 4-5
Chondroma.....	2	±	—	...	..	..	...	...	Clear	Cervical 7
B. Cerebrospinal Fluid Findings in Thoracic Group										
Sarcoma.....	...	..	..	...	..	..	2	4+	Yellow	Thoracic 5-9
Sarcoma.....	...	..	..	...	..	..	5	4+	Yellow	Thoracic 6-8
Angioma.....	...	..	..	...	..	..	1	+	Clear	Thoracic 5-7
Fibroma.....	0	0	Clear	...	..	..	...	...	..	Thoracic 10-12
Fibrosarcoma.....	...	..	..	...	..	..	2	3+	Yellow	Thoracic 11-12
Fibrosarcoma.....	...	..	..	5	0	Clear	...	...	..	Thoracic 7-8
Angioma.....	...	..	..	0	3+	Clear	...	...	..	Thoracic 1-4
Endothelioma.....	...	..	..	...	..	..	0	2+	Clear	Thoracic 2-3
Tuberculosis.....	...	..	..	...	..	..	36	3+	Clear	Thoracic 5-6
C. Cerebrospinal Fluid Findings in Cervical Group										
Melanosarcoma....	?	+	—	...	..	..	...	...	..	Cervical 6-7
Fibrosarcoma.....	0	+	—	...	..	..	...	...	..	Cervical 6-8
Fibroma.....	...	..	..	...	..	..	0	4+	Clear	Cervical 3-7
Chondroma.....	2	±	—	...	..	..	...	...	..	Cervical 6-7
Chondroma.....	...	..	..	0	±	Clear	...	...	..	Cervical 6

of this finding, the cerebrospinal fluid in fifty-two intradural extramedullary tumors was reviewed. Xanthochromia was found in thirteen of the series, or 31 per cent., while in the extradural group it was found in three of nineteen cases, or 16 per cent.

Yellow fluid was found in none of the primary vertebral tumors although a marked increase in globulin was present in two of the fluids.

In both extradural spinal tumors and intradural spinal tumors, the color of the fluid varied considerably according to the tumor level. When the block was below approximately the fifth thoracic segment, the color of the fluid was a deeper yellow than when the block was higher. The degree of xanthochromia seemed to be dependent not alone

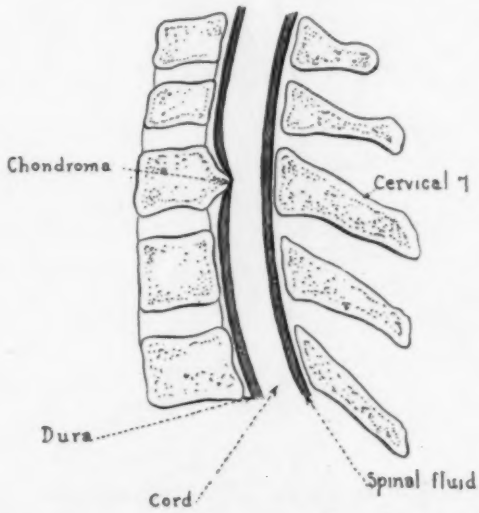

Fig. 2.—Sagittal section showing discrete pressure on the spinal cord caused by a chondroma.

on the same completeness of the block, but on the level at which this block occurred (Table 7). This is perhaps suggestive, for in high tumors the color of the fluid was described as lemon, yellow, straw or canary yellow, while the fluids of lower tumors were described as yellow, amber and greenish yellow. The segmental distribution of the tumors giving rise to xanthochromia in both the extradural and intradural series is seen in Figure 2.

The globulin content varied with the degree of pressure, and tended to be less the higher the tumor, although this was not so constant as was the variation in color of the fluid. Unfortunately, the method used to estimate the quantity of globulin was not quantitative, and strict com-

parisons cannot be made. In our more recent studies of the cerebrospinal fluid, quantitative procedures are being used, as likewise a definite color scale which permits us to make a colorimetric determination of the xanthochromia. This, we hope, will give us more accurate information.

Dr. Cushing and Dr. Ayer,[4] in their paper on xanthochromia in spinal cord tumors, differed in their opinion as to the source of the xanthochromia. Dr. Cushing was of the opinion that "the fluid changes were due to a transudation from the surface of the tumor itself," while Dr. Ayer, in agreement with Mestrezat and others, held that the xantho-

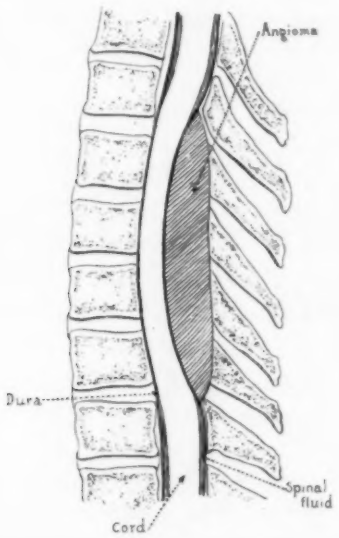

Fig. 3.—Sagittal section shows the diffuse pressure over a large number of segments caused by an angioma or a sarcoma.

chromia was due to a transudation from the obstructed veins of the spinal cord. In this series of extradural tumors, the interposition of the dura and the arachnoid between the tumor and the fluid precludes the possibility of xanthochromia being derived by transudation from the tumor into the cerebrospinal fluid, yet xanthochromia and increased globulin were found. Whatever may be the source of xanthochromia in intradural tumors, in extradural tumors transudation from the tumor into the fluid cannot be a factor in determining the presence of xanthochromia.

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4. Cushing, Harvey, and Ayer, J. B.: Xanthochromia and Increased Protein in the Spinal Fluid Above Tumors of the Cauda Equina, Arch. Neurol. & Psychiat. **10**:167 (Aug.) 1923.

The studies of Drs. Cushing and Weed [5] have shown that normally approximately 90 per cent. of the cerebrospinal fluid is absorbed over the hemisphere through the arachnoid villi into the venous sinuses, and that a small percentage may be absorbed along the nerve roots of the

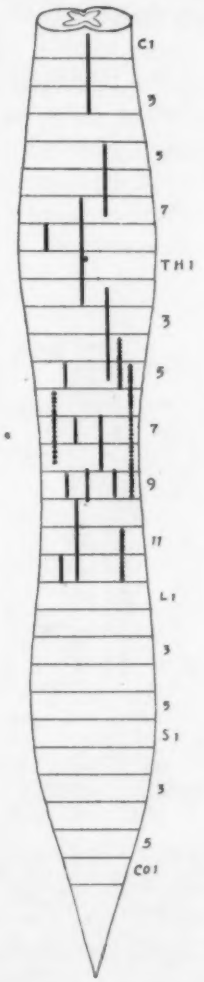

Fig. 4.—Segmental distribution of tumor giving rise to xanthochromia. Broken line indicates extradural tumors.

spinal cord. It is perhaps possible that when there is a block of the cerebrospinal fluid geater absorption of the walled-off fluid through the spinal system takes place than normally occurs. If this be true, the

5. Cushing, Harvey, and Weed, Lewis H.: Studies on the Cerebrospinal Fluid, J. M. Res. **31**, Sept., 1914.

difference might perhaps be accounted for by the composition of the cerebrospinal fluid at different levels. When the block is at a high level a greater number of spinal nerves is present and a greater degree of absorption can thus take place than when the block is at a lower level. Furthermore, it may be that the degree of absorption is proportionately greater above than below the fifth thoracic segment.

REMISSIONS AND ACUTE EXACERBATIONS

Remissions and exacerbations have been considered by some as characteristic of extradural spinal tumors. I was, however, surprised to find that when the history was carefully gone into and a thorough record made, remissions and exacerbations were not encountered. Indeed, a noteworthy gradual and progressive increase in the symptoms and course

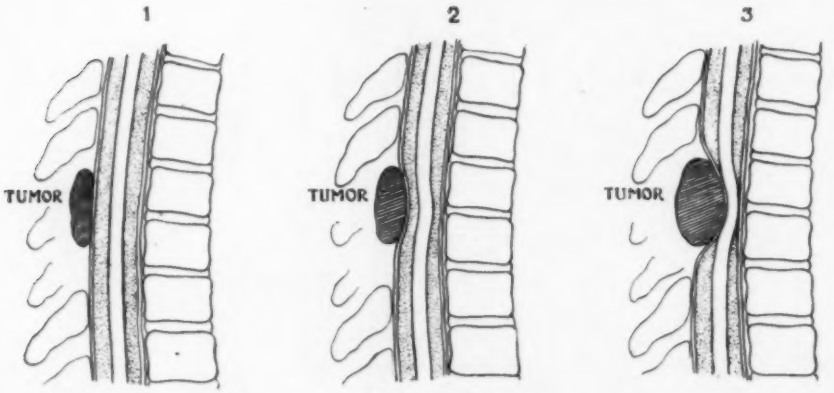

Fig. 5.—Fluid buffer between tumor and spinal cord. In No. 1 and No. 2, the pressure on the spinal cord is dispersed over a wide area by means of the noncompressible fluid covering. No. 3, tumor has grown so as to force away the fluid, and pressure is now direct on the cord.

of the disease has been found. Frequently, there was rapid progress leading to complete paralysis, with marked sensory disturbances and loss of bladder and rectal control. The symptoms preceding the sudden accentuation may have been vague and somewhat indefinite.

In intradural tumors pressure is exerted on the cord directly without the protection of the spinal fluid covering, and in this group of tumors, although they seldom attain the size common in extradural tumors, early and gradual compression of the spinal cord is more common. The symptoms and findings in this group of tumors is, therefore, more definite; the pressure is direct, not indirect, through the fluid covering. Consequently, the phase characteristic of sudden progression of symptoms due to the transition from indirect to direct compression is not encountered as a rule in intradural tumors, whereas in extradura

tumors it is commonly observed. When an extradural tumor has reached nearly sufficient size to force the fluid away and exert direct pressure on the cord, the transition from indirect to direct pressure may be brought about artificially and with great suddenness by lumbar puncture and withdrawal of the spinal fluid. In three cases of this series, lumbar puncture was followed in from twelve to twenty-four hours by marked accentuation of the symptoms and the addition of new signs. It is possible that accentuation of symptoms after lumbar puncture may occur only when the tumor has reached such a size that little further growth would exert direct pressure on the cord and give rise to a sudden accentuation of symptoms with the addition of new signs.

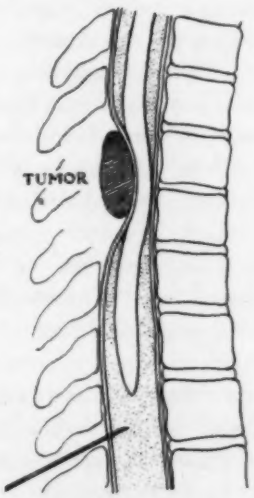

Fig. 6.—Effect of withdrawing of fluid buffer by lumbar puncture. By withdrawal of the fluid, the tumor is permitted to exert direct pressure on the spinal cord without the intermedium of the fluid buffer.

Thus the withdrawal of a relatively small amount of fluid converts this indirect pressure into direct cord pressure; in other words, there is perhaps a critical moment at which withdrawal of cerebrospinal fluid may bring about this conversion.

The influence of lumbar puncture on the symptoms and signs of spinal compression has been described in a previous paper by Dr. Elsberg and myself.[6] Lumbar puncture with withdrawal of fluid may be utilized as a diagnostic method in cases showing indefinite signs and may permit a level to be determined. When lumbar puncture is performed as an aid in diagnosis, enough fluid should be withdrawn to

6. Elsberg, Charles A., and Stookey, Byron: The Mechanical Effects of Tumors of the Spinal Cord, Arch. Neurol. & Psychiat. **8**:502 (Nov.) 1922.

permit direct pressure of the tumor on the cord and then should be followed by a comparison of the neurologic signs before and after the puncture. Occasionally additional signs will not be noted for from twenty-four to thirty-six hours.

RESULTS OF SURGICAL INTERFERENCE

The surgical end results after removal of extradural tumors is not as brilliant as in intradural tumors. In the cervical group, the tumor was removed in all instances, and improvement likewise shown in all. In two, local recurrence took place—one a melanosarcoma and the other a fibrosarcoma—although in the latter after an interval of nine years. Rapid improvement, so frequently observed in intradural tumors after removal, did not take place; recovery was slower and in some not as complete. This held true also for the tumors of the thoracic region not primarily of the vertebrae. In this group, improvement was noted in all, even though cord compression was severe. In one instance the improvement was extremely slow, yet after being bedridden for a year, this patient was able to walk. When the tumor is as extensive as in several of the patients in this group, it is possible that the delayed improvement may be due to small remains of the tumor which have not been removed. Extradural sarcomas tend to surround the spinal cord, and it is difficult to determine whether or not a ventral extension of the tumor has been left behind. After the decompressive effects of laminectomy, the spinal cord may accommodate itself to small obstructions if the parts of the tumor are left. Therefore, if complete removal is not certain, radiation, either roentgen ray or radium, is indicated.

The end results of surgical interference in primary tumors of the vertebrae vary according to the pathology. Benign tumors, such as osteochondroma and chondroma, are more or less definitely circumscribed, and following removal show considerable improvement, whereas primary sarcomas of the bone do not show improvement. Here again it is possible that deep radiation may be of some help.

It is interesting to note that in this group even temporary improvement did not follow in spite of the decompressive effect of laminectomy. This perhaps is to be expected, since interruption of cord conduction in this group did not seem to be due direcly to pressure of the tumor on the cord, but rather to interference with its blood supply.

CONCLUSIONS

1. The noncompressible fluid covering of the spinal cord causes dispersion of pressure in early stages of extradural tumors and consequently vagueness of symptoms.

2. Wide variation of pathology as found in extradural tumors gives rise to wide variation in the clinical course and signs.

3. The interval between presenting symptom and operation in extradural spinal tumors is shorter than in intradural spinal tumors.

4. Bands of hyperesthesia may be found below the level of the tumor in areas remote from the segment directly involved. Thus bands of hyperesthesia do not necessarily indicate root involvement or the level of the tumor.

5. Burning or cold sensations are associated more often with ventral than with dorsal tumors.

6. Bladder and rectal disturbances appear late in both intradural and extradural tumors, but occur at a later period in extradural tumors.

7. In extradural tumors other than primary vertebral tumors, roentgen-ray changes may be found showing either a localized enlargement of the vertebral canal or a scoliosis in the region of the tumor.

8. Changes in bony structure in primary vertebral tumors may or may not be shown by the roentgen ray.

9. Tenderness of the vertebral spines may correspond to the segment of the cord compressed or to the vertebrae beneath which the tumor lies. Tenderness of the spine is not of value as a point of differential diagnosis between intradural and extradural tumors.

10. Neither xanthochromia nor globulin is found in the cerebrospinal fluid unless marked compression of the cord exists. The depth of color varies with the level of the block, the darker shades of yellow occurring below approximately the fifth thoracic segment and the lighter shades above this level.

11. In this series of extradural tumors, xanthochromia cannot be considered due to a transudation from the tumor, since the dura and arachnoid are interposed between the tumor and the cerebrospinal fluid.

12. Neither remissions nor acute exacerbations in the clinical course of the symptoms were found. A thorough history usually revealed a steady progression.

13. Acute accentuation of the symptoms with addition of new signs may occur relatively suddenly when finally the tumor is large enough to force out the cerebrospinal buffer and exert direct pressure on the cord instead of indirect pressure through the cerebrospinal fluid.

14. Artificial conversion of the pressure from indirect to direct pressure may be made by withdrawal of the cerebrospinal fluid by lumbar puncture. When this is done, accentuation of the symptoms and addition of new signs may take place. In those patients having vague and indefinite signs, withdrawal of fluid may aid in the diagnosis of tumor and in determining the level.

15. Surgical end results on the whole are good, but not as brilliant as in intradural spinal tumors.

SENSORY DISTURBANCES IN MULTIPLE SCLEROSIS

WITH SPECIAL REFERENCE TO THE OCCURRENCE OF LEVEL LESIONS AND THEIR DIFFERENTIATION FROM THOSE OF INTRAVERTEBRAL TUMORS *

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The first reference in neurologic literature to the occurrence of disturbances of sensation in multiple sclerosis is found in the report of a case of this disease by Cruveilhier. The latter, writing in 1835, describes a case which he calls "induration of the cord with paraplegia," and states that he found "in places the sensibility diminished and delayed." Since then numerous observers have reported sensory disturbances in multiple sclerosis, so that their occurrence in this disease is now a generally admitted fact. Some writers have even gone so far as to state, and correctly so, that subjective sensory disturbances are among the most characteristic of the early signs of the disease. Objective sensory disturbances, on the other hand, have not been given the attention they deserve in the symptomatology and differential diagnosis. Most textbooks mention the presence of objective sensory disturbances, and dismiss the subject with a trite statement that they are present in a large number of cases, but are patchy in distribution and vague and variable in nature.

Our aim is to point out the important rôle objective sensory disturbances may play in the clinical picture of multiple sclerosis, and the difficulty which their presence adds to the differential diagnosis between this disease and localized cord lesions. With this view in mind, we have reviewed and critically analyzed all the cases of multiple sclerosis admitted to the neurologic service of the Mount Sinai Hospital from January, 1920, to the date of writing.

Our clinical material consists of forty-four cases; twenty-two males and twenty-two females. The average age of the patients at the time of the onset was 28 years, the youngest being 6 years and the oldest 47. In twenty-nine of these, the clinical diagnosis of multiple sclerosis could readily be established on admission to the hospital; in the remaining fifteen, the history of onset and the chronologic order of the appearance of symptoms was so indefinite, and the evidence of cranial nerve

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involvement so equivocal, that the diagnosis was rendered extremely difficult. Special difficulties were encountered in the cases in which the objective sensory disturbances, unlike those found in some of the other twenty-nine cases, were permanent in nature and assumed a definite segmental type of distribution. Many of these were indicative of level lesions, and, when taken in conjunction with the motor symptoms, presented an atypical, as well as typical, Brown-Séquard syndrome. It was in these cases that a positive diagnosis could not be made until, after prolonged observation, the cranial nerve symptoms became more definite, or until one or more remissions had occurred, whereas in four cases the differential diagnosis between multiple sclerosis and spinal cord tumor was so difficult that exploratory laminectomy was deemed advisable.

An analysis of the sensory disturbances in our material shows that thirty-five patients (79.54 per cent.) presented some evidences of disturbed sensibility. Subjective sensory disturbances, such as, numbness, tingling, burning, itching, a feeling of heaviness, stiffness or pressure in one or more limbs, in the trunk or head were found in twenty-eight patients (63.63 per cent.). Objective sensory disturbances referable to definite sensory systems with definite anatomic distribution were found in twenty-four patients (54.54 per cent.). Ten presented *subjective* disturbances *only* (22.72 per cent.); six, *objective* disturbances *only* (13.63 per cent.); seventeen, *both objective and subjective* (38.63 per cent), and nine had *neither subjective nor objective* sensory disturbances (20.45 per cent.).

The sensory systems involved were as follows: Of the six patients with *objective* disturbances *only*, two had signs referable to the spinothalamic tracts, whereas all of them presented evidences of posterior column involvement. Four patients in this group showed definite sensory levels; two had astereognosis. There was no vertebral tenderness in any of the six cases. Of the seventeen with *both objective and subjective* disturbances, sixteen showed affection of the spinothalamic tracts; ten, posterior column involvement; eleven, definite sensory levels; one, astereognosis; seven, vertebral tenderness. Ten had posterior column involvement together with affection of the spinothalamic tracts, whereas one had only posterior column involvement.

At this point we wish to call attention to the fact that of the twenty-four patients showing objective sensory disturbances, sixteen had spinothalamic involvement, and twelve had evidences of posterior column disturbances. The greatest number of spinothalamic disturbances occurred in the cases presenting both objective and subjective disturbances. It was also striking that in the entire series, all cases showing objective sensory disturbances had marked symptoms referable to the pyramidal tracts. This was especially the case in the patients presenting

objective sensory disturbances *only*. There was apparently no statistical relationship between the frequency of posterior column involvement and that of the pyramidal tracts. It may be said, in general, that the severity of the spinothalamic symptoms ran parallel to that of the motor (pyramidal tract) symptoms.

As to the individual types of sensation involved, it was noticeable that the sense of pain was affected most commonly, next that of touch, and least of all that of temperature. Vertebral tenderness was more noticeable in the cases showing spinothalamic involvement than in those

TABLE 1.—*Cases in Which History, Course of Disease and Cranial Nerve Symptoms Definitely Established the Clinical Diagnosis of Multiple Sclerosis*

	Case	Subjective	Objective	Spinothalamic Tracts: Pain	Spinothalamic Tracts: Touch	Spinothalamic Tracts: Temp.	Posterior Columns: Vibratory	Posterior Columns: Joint Sense	Posterior Columns: Astereognosis	Vertebrae Tenderness	Sensory Level	Remarks
1	R. R.	+	..	..	..	..	..	..	..	..	..	
2	R. K.	..	..	..	..	..	..	..	..	..	..	
3	P. F.	+	..	..	..	..	..	..	..	..	..	
4	O. O.	+	..	..	..	..	..	..	..	..	..	
5	J. C.	+	..	..	..	..	..	..	..	+	..	
6	J. K.	..	..	..	..	..	..	..	..	..	..	
7	S. S.	..	..	..	..	..	..	..	..	..	..	
8	M. I.	..	..	..	..	..	..	..	..	..	..	
9	C. L.	+	..	..	..	..	..	..	..	..	..	
10	D. S.	..	+	..	..	..	+	+	..	..	..	3 admissions
11	T. J.	..	+	..	..	..	..	+	+	..	..	
12	M. S.	+	..	..	..	..	..	..	..	..	..	
13	I. T.	+	..	..	..	..	..	..	..	..	..	
14	G. W.	+	..	..	..	..	..	..	..	..	..	
15	B. L.	..	..	..	..	..	..	..	..	..	..	
16	A. M.	..	..	..	..	..	..	..	..	..	..	
17	M. L.	..	..	..	..	..	..	..	..	..	..	
18	S. W.	+	..	..	..	..	..	..	..	..	..	
19	H. F.	+	+	..	..	..	+	+	+	..	..	
20	B. H.	+	+	?	+	+	..	..	..	..	+	
21	L. E.	+	+	..	..	..	+	..	..	..	+	
22	N. R.	..	+	..	..	..	+	..	..	..	..	
23	M. C.	..	..	..	..	..	..	..	..	..	..	
24	P. R.	..	..	..	..	..	..	..	..	..	..	
25	H. M.	+	..	..	..	..	..	..	..	..	..	
26	S. N.	..	+	..	..	..	+	..	..	..	..	
27	R. V. S.	..	+	..	..	..	+	..	..	..	..	
28	A. K.	..	+	..	..	..	+	..	..	..	+	
29	B. F.	..	..	..	..	..	..	..	..	..	..	
Total		14	9	1?	1	1	7	3	2	1	3	

+ = present.

showing only affection of the posterior columns. The vibratory sense seems to have been involved more frequently than joint sensibility. The duration and progress of the disease apparently bore no relation to the presence of sensory disturbances.

In comparing our own statistics (Tables 1, 2, 3 and 4) as to the frequency of involvement of the spinothalamic tracts and posterior columns, respectively, with those in the literature, we were struck by the marked variation in the findings as reported by different observers. Most authors make a special point of the fact that posterior column involvement in multiple sclerosis is far more frequent than that of the

TABLE 2.—*Cases in Which Sensory Symptoms Were so Marked and Cranial Nerve Symptoms so Slight That the Diagnosis of Multiple Sclerosis Could Not Be Made with Certainty Until the Patients Had Been Under Prolonged Observation* *

	Case	Subjective	Objective	Spinothalamic Tracts: Pain	Touch	Temp.	Posterior Columns: Vibratory	Joint Sense	Astereognosis	Vertebrae Tenderness	Sensory Level	Remarks
1	S. H.	+	+	+	+	+	..	..	..	..	+	
2	M. J.	+	+	+	+	+	..	..	..	..	+	3 admissions
3	P. G.	+	+	+	+	..	..	..	..	+	B.S. +	3 admissions
4	M. H.	+	+	+	+	+	..	..	..	..	..	
5	R. H.	+	+	+	+	?	..	..	..	..	B.S. +	2 admissions
6	P. T.	..	+	+	..	..	+	..	+	..	?	
7	K. P.	+	+	+	+	+	+	..	..	..	+	
8	J. G.	+	+	+	+	..	+	+	..	+	+	Laminectomy
9	D. K.	+	+	+	+	..	+	+	..	?	+	3 admissions
10	H. F.	+	+	+	..	..	..	..	..	+	?	
11	C. P.	+	+	+	+	+	+	..	..	+	+	Laminectomy
12	M. B.	+	+	+	..	..	+	..	..	..	..	
13	L. F.	+	+	+	+	+	+	+	..	+	+	2 laminectomies
14	C. S.	+	+	+	+	?	+	+	..	+	+	3 admissions
15	A. P.	+	+	+	+	+	+	+	..	..	B.S. +	Laminectomy
	Totals	14	15	15	12	7	9	5	1	7	13	5 laminectomies

B.S. = Brown-Séquard syndrome; + = present.

* The question as to the presence of spinal cord tumor came up constantly for consideration. Four of these patients had to be subjected to exploratory laminectomy (one patient had to be laminectomized twice) to exclude the presence of a tumor.

TABLE 3.—*Statistical Summary of Sensory Disturbances*

		Per Cent.
Total number of cases studied	44	
Males	22	
Females	22	
Average age at onset of disease	28 years	
Youngest patient's age	6 years	
Oldest patient's age	47 years	
	Cases	
Definite clinical picture of multiple sclerosis on admission	29	
Question as to whether case was one of multiple sclerosis or tumor	15	
Evidences of disturbed sensibility, subjective, objective or both	35	79.54
Entire absence of any sensory disturbances	9	20.45
Subjective sensory disturbances found	28	63.63
Subjective sensory disturbances only	10	22.72
Objective sensory disturbances found	24	54.54
Subjective and objective sensory disturbances in the same patient	17	38.63
Objective sensory disturbances only	6	13.63

TABLE 4.—*Analysis of Types of Sensibility Involved*

	Cases
In the group of cases with objective sensory disturbances only:	
Spinothalamic involvement	2
Posterior column involvement	6
Astereognosis	2
Sensory levels	4
Vertebral tenderness	0
In the group of cases with both objective and subjective sensory disturbances:	
Spinothalamic involvement	16
Posterior column involvement	10
Astereognosis	1
Sensory levels	11
Vertebral tenderness	7

spinothalamic tracts. Birley and Dudgen in studying thirty-five cases of multiple sclerosis found disturbances in deep sensibility, especially vibration, in 65.7 per cent., while disturbances of cutaneous sensibility were found in 33.3 per cent. They make no special mention of sensory levels. Sachs and Friedman in a study of 141 cases found the posterior columns affected in 17 per cent. and pain, touch and temperature disturbances in 16 per cent.; among the latter they found cases with zones of hyperalgesia; they also found astereognosis in five of their cases. We merely cite these two series of statistics to show the great discrepancies in the figures of these two groups of well-known observers. In studying the older literature on this phase of the subject, one encounters still greater discrepancies.

This is not a purely academic problem. It is one of practical value, with the importance of which we were greatly impressed when in eight of our cases the presence of a spinal cord tumor was seriously entertained. In almost all of these there were sufficient indications to advise laminectomy, and in four cases this operation was actually performed, one of the patients having been subjected to laminectomy twice. Here we wish to cite four cases.

REPORT OF CASES

CASE 1.—J. G., aged 30, a married American woman, was admitted to the hospital on June 29, 1922, with the history that seven years ago she began to have dizzy spells with a tendency to fall to either side when in the dark. This has persisted up to the present time. Two years ago, she developed stiffness in both legs; this is becoming progressively worse. One year ago, she is said to have seen "double" and had a squint. During the last two years, she has been having delayed and interrupted urination and constipation.

The positive findings on admission were: ovoid pupils, nystagmoid movements to the right and left, right facial weakness (?) and a suggestion of a head tremor. The deep reflexes of the upper extremities were active, and those of the lower, hyperactive but equal. There were bilateral patellar and ankle clonus and a bilateral Babinski sign. Both lower limbs were extremely spastic. The vibratory sense was lost from the toes to the waist line. Sense of position was disturbed in the right foot. There was a definite belt of hyperesthesia from the fourth to the seventh dorsal segments on the right side and from the sixth to the ninth dorsal on the left, and a zone of hyperalgesia from the fourth to the sixth dorsal on both sides. No ataxia was present. There was percussion tenderness over the second dorsal vertebral spine. Four days after admission, lumbar puncture yielded a clear fluid with 10 lymphocytes and no globulin. A positive Queckensted reaction was obtained (no increase in pressure of the spinal fluid as seen by the manometer on compression of the jugular veins). The blood and the cerebrospinal fluid Wassermann tests were negative. After lumbar puncture, vibration was felt better on the right than on the left (below the seventh dorsal); sense of position of toes was faulty on both sides; a definite zone of hypalgesia extended from the original zone of hyperalgesia down to the left thigh. Another sensory examination three days later revealed hypalgesia in the distribution of the ninth and tenth dorsal and also the left perianal region; vibratory and joint sensibility was disturbed in both lower

extremities; there was percussion tenderness of the spine between the third and sixth dorsal. No change occurred in the cranial nerve and motor symptoms. The patient was presented before the conference of the neurologic division of the hospital, and the consensus of opinion was that the most likely diagnosis was multiple sclerosis with a pseudotumor syndrome, and laminectomy was advised to exclude a possible cord tumor. Eighteen days after admission, Dr. Elsberg performed an exploratory laminectomy; the first, second and third dorsal laminae were removed, and on incising the dura, an accumulation of fluid and a flattened cord were found, but no neoplasm. A sensory examination four days after operation revealed a belt of hyperesthesia on the left side between the first and second dorsal and a similar belt on the right side between the second dorsal and second cervical, with no changes in the other sensations from those noted previously. She was discharged from the hospital on Aug. 30, 1922, with some subjective improvement and less spasticity, but with practically the same objective sensory disturbances as she showed a week after laminectomy. Subsequent observation of this case left no doubt that the patient was suffering from multiple sclerosis. It may perhaps be of interest to add that at no time did this patient show any evidences of disturbances of thermal sensibility, whereas all other forms of sensibility were disturbed at one time or another.

Case 2.—L. F., a woman, aged 32, married, was admitted to the hospital on Dec. 17, 1917, with the history that in 1912 she began to have numbness and a sensation of "pins and needles" in the fingers of both hands. Soon thereafter she developed similar sensations from the waist downward, which was followed by a burning pain in the back of the neck. Shortly afterward she experienced stiffness of both lower extremities, first of the right and then of the left leg, and she began to stagger and to fall frequently. She never lost consciousness, and never had vertigo. At the same time she had difficulty in starting urination, and became constipated. Six months after the onset of these symptoms, she was operated on several times for an ischiorectal abscess and fistula in ano; after one of these operations, she developed many furuncles throughout the body. Two months before admission, she began to experience a girdle sensation about the chest, with numbness in the fingers of both hands. For the past three or four months, she had had urinary incontinence at night and every hour during the day; occasionally she would have dribbling of urine. Examination on admission showed diminished movements of the right hip joint and some interference with the movements of the left hip joint. Both lower limbs were spastic, the right more than the left. Both knee reflexes were markedly exaggerated, the right more than the left; bilateral patellar clonus and a bilateral Babinski sign were present. Owing to marked spasticity of the gastrocnemii group, the Achilles reflex could not be elicited on either side. The lower left abdominal reflex was present, the others could not be obtained. The seventh and eighth dorsal spines were tender to percussion. Sensory examination showed a slight diminution to touch below the second dorsal; a zone of hyperalgesia between the second cervical and the second dorsal, with a diminution of pain sensibility below this level; thermal sensibility was increased from the sixth dorsal to the second cervical in front. All laboratory findings were negative. An exploratory laminectomy was advised and performed by Dr. Elsberg on Jan. 2, 1918; the first, second and third thoracic laminae were removed. The operative diagnosis was localized meningomyelitis; no tumor was found. She made an uneventful postoperative recovery, and within a few weeks she had improved sufficiently to enable her to do some housework. She could walk with ease, had regained the control of her sphincters and suffered from no pain.

She remained apparently well until July, 1918, when she was admitted to the Montefiore Hospital with the history that all her symptoms had returned suddenly. She had great difficulty in walking, a girdle sensation about her chest, shooting pains in the legs and vesical and rectal incontinence. Examination showed a complete spastic paraplegia, with complete loss of deep sensibility in both feet; there was a belt of hyperesthesia and hyperalgesia corresponding to the fifth, sixth and seventh dorsal segments on the right side. Abdominal reflexes were absent on the right side and present on the left. She remained practically in this condition for four months, when her zone of hyperalgesia and hyperesthesia was found to extend down to the twelfth dorsal segment. At no time was there any evidence of cranial nerve involvement.

On Nov. 23, 1921, she was readmitted to the Neurologic Service of the Mount Sinai Hospital, where she showed marked spasticity, weakness and exaggerated deep reflexes in both lower extremities, more marked on the right than on the left, bilateral ankle clonus and Babinski sign, absent abdominal reflexes, but no cranial nerve symptoms. Sensory examination revealed pain, touch and temperature sense diminished up to the third cervical, more on the left than on the right, most marked (especially temperature) at the fourth cervical, complete loss of postural sense in both lower extremities and loss of vibratory sense from the toes to the free costal margins. Laminectomy was advised and performed. After removal of the laminae of the fourth, fifth, sixth and seventh cervical, the dura was incised; this was followed by an escape of a large amount of cerebrospinal fluid under increased pressure; the cord was found to be flattened, but no tumor was found. She was discharged from the hospital on March 16, 1922, with very little change in the signs and symptoms.

She remained in this condition until Nov. 1, 1922, when she was readmitted to the Montefiore Hospital. Examination revealed the left pupil larger than the right, bilateral nystagmus in both lateral planes, right temporal pallor, perioral tremor, ptosis of the fingers of the outstretched hands, some ataxic tremor of the left hand, disturbances of posture in the right hand and fingers, no astereognosis, poor right grip, absent abdominal reflexes and bilateral pyramidal tract involvement, indefinite disturbances in pain sensibility on the entire left side of the body from the toes up to the fourth dorsal, and definite disturbances of posterior column sensibility up to the fourth dorsal. There was apparently no disturbance in touch and temperature, and no level of hyperesthesia or hyperalgesia. She could hold her urine but had no control of her rectal sphincter. All the laboratory findings were repeatedly negative both at Mt. Sinai and at Montefiore Hospital.

She has been under continuous observation by one of us (Keschner). The notes on her examination just prior to the writing of this paper state that there is a definite progression of all her cranial nerve and motor symptoms. Her sensory disturbances remain stationary. She undoubtedly has a case of advanced multiple sclerosis.

Case 3.—An Austrian woman, aged 26, single, was admitted to the hospital on Aug. 6, 1923. Her present illness began in February, 1922, with weakness in both legs, dizziness and pain in the middle of her spine. These symptoms were gradual in their onset and did not incapacitate her from attending to her work, which was that of a milliner. In May, 1922, she had to give up her work, because she could not walk without support. In January, 1923, she began to have sharp pains in the upper dorsal vertebrae; these pains were especially severe when she remained in the recumbent posture. During this period she also had paresthesias with a sense of compression in the toes and legs. In June,

1923, she developed pain in the shoulders, radiating down to the fingers, which began to feel numb and "clumsy." Ever since the onset, she has had occasional frequency of urination; for the past three months, urgency, and for the past two weeks hesitancy. Neurologic examination revealed: the left pupil perhaps larger than the right; both reacted somewhat sluggishly to light, but better on accommodation; right facial asymmetry and a questionable left corneal hypesthesia. The tendon and deep reflexes in the upper extremities were active and equal; those of the lower extremities hyperactive, but equally so; there were bilateral ankle clonus and a bilateral Babinski sign. Abdominal reflexes could not be elicited. There were marked weakness and spasticity of both lower extremities and some ataxia on performing the heel to knee test on both sides, but no ataxia in the upper extremities. Her gait was spastic, and she could not stand without support. Sensory examination revealed a zone of hyperalgesia from the third to the sixth dorsal segments both anteriorly and posteriorly; below this level she was slightly hypalgesic down to the toes. There was also some impairment in heat and cold perception in the lower extremities (patchy in distribution); vibratory sense was lost from the toes up to the sixth dorsal; sense of position was undisturbed. There was definite vertebral tenderness on percussing the second dorsal spine. The ophthalmologist reported evidences of slight optic neuritis in both disks, not typical of multiple sclerosis and normal visual fields. Lumbar puncture yielded a clear fluid under normal pressure; there were 6 lymphocytes to the cubic millimeter. The Queckensted test was normal. Blood and spinal fluid Wassermann tests were negative; there was no globulin, and the colloidal gold curve was normal. All other laboratory findings, including roentgen-ray studies, were negative. There was no change in the sensory findings after lumbar puncture. Eleven days after admission she was subjected to an exploratory laminectomy by Dr. Neuhof. The laminae of the second, third, fourth and fifth dorsals were removed, and the dura was incised. No neoplasm was found; probing upward and downward revealed no obstruction. The cord appeared normal. Four days after laminectomy, she developed pain and numbness over the ulnar distribution of the right forearm, with hyperalgesia over the same area, including the ulnar distribution of the right hand. Two days later, she developed a sensation of "rings" around the fourth and fifth fingers of the right hand with a distinct hyperalgesia over the entire distribution of the eighth cervical and the first dorsal on the same side. All these sensory disturbances (subjective as well as objective) had completely disappeared on the eighth day after operation. On her discharge from the hospital two weeks later, the neurologic findings, both motor and sensory, were the same as on admission.

We wish to point out here that fifteen months before admission this patient was seen by one of us (Keschner); at that time the motor signs and subjective symptoms were practically the same as those noted on admission to the hospital, except that there were absolutely no evidences of cranial nerve involvement and no objective sensory disturbances. The further history of this case leaves no doubt that this patient is suffering from multiple sclerosis.

Case 4.—A. P., a Russian, aged 34, a tailor, was admitted to the hospital on Nov. 30, 1917, complaining of inability to walk for the last three months. His illness began four years before, with shooting pains and slight weakness and stiffness in the left leg. These symptoms were gradually progressive until three months ago, when both of his legs became stiff and paralyzed; this was associated with "sticking" pains in the left hypochondrium with an occasional feeling of numbness in this area. One and a half weeks before admission to the

hospital, he suddenly developed urinary incontinence. Examination on admission revealed a questionable nystagmus in the extreme lateral gaze, spastic paraplegia with evidences of bilateral pyramidal tract involvement in both lower limbs, absent abdominal reflexes, a level of hyperesthesia and hyperalgesia between the sixth and seventh dorsal segments with analgesia and thermoanesthesia from the toes up to the ninth dorsal. Lumbar puncture on Dec. 2, 1917, yielded a fluid under normal pressure, with slightly increased globulin; the Wassermann test was negative. On the day following lumbar puncture, the level of hyperesthesia and hyperalgesia was found to have extended up to the fifth dorsal, and he developed incontinence of feces. He was transferred to the neurologic service, with a diagnosis of spinal cord tumor or multiple sclerosis (?), and he was subjected to an exploratory laminectomy at the level indicated. At operation, Dr. Elsberg found a localized adhesive meningitis and arachnitis. Two weeks after operation, examination showed no changes. Following the operation, he stayed for one year at the Montefiore Hospital, where he began to regain power in his legs so that he could walk with the aid of a cane; his bladder and rectal control had also returned. He was quite comfortable until January, 1923, when his left leg became weak again, and he began to have pain in the left thigh, and developed a tremor of the same limb. He remained in this condition until July 27, 1923, when he was readmitted to the Mount Sinai Hospital. Examination at this time revealed a questionable nystagmus (bilateral), a right central facial weakness, marked weakness and spasticity of both lower extremities, of the left more than the right; knee and ankle reflexes exaggerated, the left more than the right; left ankle clonus and a bilateral Babinski sign; the abdominal reflexes were unobtainable on the left side. His gait was that of a spastic paraparesis. Sensory examination revealed loss of pain, touch and temperature on the right side from the toes up to the eleventh dorsal, and a diminution of these sensations up to the seventh dorsal and a slight diminution of touch, pain and temperature up to the seventh dorsal on the left side. Sense of position and vibration was completely lost on both sides from the toes up to the seventh dorsal. All laboratory findings, including roentgen-ray studies, were negative. His condition remained stationary, and he was discharged on Aug. 8, 1923, with the diagnosis of multiple sclerosis (?) with a Brown-Séquard syndrome. He has recently been examined by another member of the attending neurologic staff, who reports his condition to be the same as it was on his discharge from the hospital (ten months ago).

COMMENT

We refrain from making any epicritic remarks on these four cases, because we are well aware that every neurologist reading these reports will, in retrospect, find some logical explanation for the "wrong" diagnoses made. Analyzing a case in retrospect is of course entirely different from meeting the diagnostic problem presented at the bedside.

These cases are merely cited in corroboration of the point made long ago by Bruns and others that in some instances the pathologic process of multiple sclerosis may be such that the clinical picture resembles that seen in the Brown-Séquard syndrome. The pathologic process may involve the posterior nerve roots, giving rise to typical obstinate root pains and girdle sensations; or it may involve peripheral nerves and secondarily even the mesodermal structures, giving rise to a meningo-

myelitic syndrome (Hassin). It is just in these, the so-called spinal types of multiple sclerosis—in their early stages—in the absence of unequivocal cranial nerve signs, that sensory disturbances at definite levels make the differentiation between multiple sclerosis and intravertebral tumor difficult. The differential diagnosis between multiple sclerosis and multiple spinal tumors which run their clinical course, as they may, without root pains, may for a long period of time be absolutely impossible.

Flatau attempts to establish certain criteria for distinguishing multiple sclerosis from spinal tumors. The first of these is that multiple sclerosis is not so progressive a disease as tumor. Some of the cases of chronic multiple sclerosis remain stationary for years, whereas in intravertebral tumors there seems to be a steady progression of symptoms, although even here remissions may occasionally occur. These, however, are not of so long a duration nor so complete as those in multiple sclerosis. Second, the pains of multiple sclerosis are rarely as severe as those of tumor. Careful questioning will reveal that the pains in multiple sclerosis are more in the nature of paresthesias, whereas the pains in tumor are more in the nature of a tearing, boring or burning. The former are seldom severe enough to confine the patient to bed. Third, the objective sensory disturbances in multiple sclerosis are only rarely those of a total loss of sensation but are rather those of of diminution of sensation. "The hypesthesias," Flatau states, "he could never find to lead to a total anesthesia as is the case in the late stages of meningeal tumors."

Our cases prove the correctness of Flatau's first two differentiating criteria but do not bear out the correctness of the third. In our doubtful cases, we found below the level of hyperalgesia or hyperesthesia, a definite total anesthesia in three cases, total analgesia in two and total thermo-anesthesia in two cases; what is equally important, the total loss of these various sensations occurred in these cases early in the disease, and were permanent.

In this connection we would add that the pains in multiple sclerosis are usually not affected by change of posture, extreme extension or flexion of the head or limbs, sneezing or coughing, as is so often the case in the early stages of spinal tumors, when the pains are due to stretching of the posterior nerve roots.

In the dubious cases with level sensory disturbances involving the trunk, we were greatly aided by a careful study of the extent of the level. Great discrepancies in the extent of the level anteriorly as compared with that posteriorly would be more in favor of multiple sclerosis than of tumor. The presence or absence of root pains or of a zone of hyperesthesia or of hyperalgesia above the level of anesthesia or analgesia could not be utilized as a diagnostic point, because we have, in our

experience, encountered some cases of intramedullary disease without as well as with such zones. Similarly, we have seen extramedullary tumors that have passed the period of the so-called first phase of spinal tumor, and cases in which the tumor occupied the lateral aspect of the cord, with sensory disturbances merely those of tract compression and not of root irritation. In both of these instances, as far as we could determine, the tumors ran their course with little or no pain.

In attempting to correlate the motor and sensory findings in this differentiation, we were greatly aided by bearing in mind the well-known general rule that in cases in which the cord is being gradually compressed from without by progressive disease of the meninges or vertebrae, there is usually a progressive paraplegia with changes in the deep and superficial reflexes. Here anesthesia appears late in the disease, being preceded by subjective sensory disturbances, after which hypesthesia develops, and last of all, anesthesia. Moreover, in compression paraplegia, the lower sacral areas are usually less deeply anesthetic than the areas above. The appearance of irritative phenomena such as pain and spasm after the appearance of the paralytic symptoms, was of no diagnostic value, because this is a common condition in intramedullary tumors which begin in the center of the cord and extend to the periphery and into the posterior root zones. One can readily conceive that a patch of sclerosis within the cord may have a similar effect. The mere presence or absence of pain in these cases is not to be relied on to prove that the condition is intramedullary or extramedullary. We have repeatedly seen intramedullary tumors run their course with pain and extramedullary tumors without pain. Of course, all these well-known facts are to be utilized diagnostically only in conjunction with all other symptoms when the patient can be kept under continuous and prolonged observation with repeated painstaking sensory examinations, preferably by the same examiner.

The finding of sensory disturbances referable to segments anatomically remote from those representing the motor symptoms, is in favor of multiple sclerosis. The lapse of an unusually long period of time between the appearance of motor and sensory symptoms is also in favor of multiple sclerosis. Localized muscular atrophies are rare in multiple sclerosis; their presence in association with sensory disturbances in the same segmental distribution speaks in favor of tumor rather than multiple sclerosis.

In the diagnostic evaluation of the cranial nerve symptoms in these doubtful cases, it must be borne in mind that in high cervical growths the ascending fibers of the trigeminal nerve may be involved. Irritation of the ciliospinal centers may give rise to dilatation of the pupil, widening of the palpebral fissure and exophthalmos on the same side as the tumor. The prolongation of this irritation will be followed by miosis

and enophthalmos. Nystagmus and even papilledema have also been observed in these high tumors. Sexual and sphincteric symptoms may also appear early in some of these cases.

Considerable diagnostic information may be obtained from lumbar puncture in these doubtful cases. A spinal fluid of normal appearance obtained under normal or low pressure with a slight lymphocytosis, normal, or slightly increased protein content, and an absence or only slight trace of globulin, in the presence of a paretic colloidal gold curve, though not conclusive, is in favor of multiple sclerosis, whereas a marked increase of globulin, xanthochromia and cloudiness (Nonne-Froin syndrome) would point to tumor. The presence of a positive Queckensted reaction, even in the absence of xanthochromia would also point to the diagnosis of neoplasm. In cases with an indefinite sensory level, the appearance of a definite level after lumbar puncture or the appearance of a level where none had existed before the puncture, would speak in favor of a tumor, whereas in multiple sclerosis there is, as a rule, no perceptible change observed in the objective sensory findings following lumbar puncture.

The presence of vertebral tenderness, ordinarily a valuable symptom in spinal tumor, did not aid us in the differential diagnosis between these two conditions. Eight of our cases of multiple sclerosis showed definite vertebral tenderness within the area of objective sensory disturbances. Roentgenograms of the vertebral column were of no value in the differential diagnosis. Injections of air or iodipin into the subarachnoid space, followed by roentgenographic studies, have recently been reported as extremely helpful in the diagnosis and localization of compressing lesions of the cord. Unfortunately, we had no opportunity to subject our patients to air or iodipin injections, and therefore have no personal experience with them. In spite of the more recent conflicting reports as to the usefulness of these methods, it would seem that after their technic has been perfected, they might be of great value in the very type of cases under consideration. Double puncture, cistern and lumbar (Ayer), would, from all reports, also seem to be a rational and valuable method in differentiating multiple sclerosis from possible tumor, but here too we cannot speak from personal experience, because none of our patients in this series was subjected to this procedure. The absence of trophic and vasomotor disturbances in the paralyzed limbs in these doubtful cases is in favor of multiple sclerosis rather than tumor.

Taking all the foregoing facts into consideration, it can readily be seen that in the present state of our knowledge there still remain some cases on the borderline between these two conditions in which a diagnosis in vivo cannot be made with any reasonable degree of certainty. It is in these cases that we do not hesitate to urge the necessity of exploratory laminectomy. We feel that by doing so, not only do we

give the patient the benefit of the doubt as regards diagnosis, but we also give him an opportunity to avail himself of the beneficial results, from a therapeutic standpoint, following laminectomy. Elsberg and Bailey were the first to show that surgical exposure of the spinal cord, even in the absence of increased intradural pressure, may and often does effect some change, which temporarily or permanently benefits or checks the symptoms of local spinal cord disease. Numerous observers have since then reported similar results, and this has also been our personal experience with two cases in this series, although we must admit that we cannot state whether the improvement was due to a remission or to the effects of the laminectomy.

An exploratory laminectomy by an experienced surgeon performed under local anesthesia is free from danger. It offers so many diagnostic and therapeutic advantages that in doubtful cases we are inclined to advise this operation early. We feel that by doing this we subject the patient to hardly any risk, and we give him the opportunity, in case his symptoms are due to tumor, to get rid of it before the latter has irreparably damaged the cord.

VENTRICULAR HEMORRHAGE WITH RECOVERY*

REPORT OF A CASE

BERNARD J. ALPERS, M.D.

PHILADELPHIA

Hemorrhage into the ventricles of the brain, while usually conceded to be fatal, need not necessarily result in death, as is shown by the case which is reported in this paper. Most writers agree that primary hemorrhage into the cerebral ventricles is both rare and fatal, and indeed that hemorrhage into the cerebral ventricles, whether primary or secondary, is always fatal. Among those who hold this view are Oppenheim,[1] Osler,[2] Church and Peterson,[3] Jones[4] and Hughlings Jackson.[5] Oppenheim[1] is of the opinion that in both primary and secondary hemorrhage "death almost inevitably occurs—indeed within the first twenty-four hours." Osler[2] states that hemorrhages which rupture into the ventricles "rapidly prove fatal," and Church and Peterson[3] state that "indications of ventricular flooding practically mean death." Jones[4] analyzed fifty-five cases of ventricular hemorrhage, and concluded that 60 per cent. of the patients die during the first twenty-four hours, and 90 per cent. during the first week. Spiller,[6] in a study of thirteen cases of ventricular hemorrhage, found that hemorrhage into the ventricles is more rapidly fatal than where the ventricles escape, but that "a moderate hemorrhagic exudate into the ventricles is not necessarily rapidly fatal." In nine of his thirteen cases in which rupture occurred into the lateral ventricles, the patients lived from twenty-four hours to sixteen days after the hemorrhage occurred. Sanders,[5] in a study of seventy cases of primary ventricular hemorrhage, found that 65 per cent. of the patients died in twenty-four hours, and that of these all but three died in twelve hours. His conclusion was that primary ventricular hemor-

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*From the Philadelphia Orthopedic Hospital and Infirmary for Nervous Diseases, service of Dr. T. H. Weisenburg.

1. Oppenheim, H.: Lehrbuch der Nervenkrankheiten, Berlin, S. Karger, 1923.
2. Osler, W., and McCrae, T.: Principles and Practice of Medicine, Ed. 9, New York, D. Appleton & Co., 1920, p. 984-992.
3. Church, A., and Peterson, F.: Nervous and Mental Diseases, Philadelphia, W. B. Saunders Company, 1906.
4. Jones, A. Ernest: The Onset of Hemiplegia in Vascular Lesions, Brain **28**:527, 1905.
5. Jackson, Hughlings, in Sanders, Edward: A Study of Primary, Immediate, or Direct Hemorrhage into the Ventricles of the Brain, Am. J. Med. Sc. **82**:85 (July) 1885.
6. Spiller, W. G.: The Duration of Life After Extensive Hemorrhage of the Brain, J. A. M. A. **51**:2101 (Dec. 19) 1908.

rhage is almost always fatal. Gordon [7] reports twelve cases of ventricular hemorrhage, seven of them primary and five secondary, in all of which the patients died within twenty-four hours; and Allen [8] reports eight cases in which no patient recovered from ventricular hemorrhage.

There are also numerous reports of isolated cases of ventricular hemorrhage, confirmed by necropsy, in which the hemorrhage was rapidly fatal. Such cases are reported by Tilney and Casamajor,[9] Lavastine and Bloch,[10] Looten,[11] Dupré,[12] Froin and Laederich.[13] Death occurred in thirteen hours in the case reported by Tilney and Casamajor,[9] in seven days in the cases of Lavastine [10] and Froin,[13] in ten days in the case of Looten,[11] and in thirteen days in Dupré's [12] case. Similar cases are reported by Abeille,[14] Robert,[15] Royster,[16] Sauvan,[17] Ludlum,[18] and Pope.[19] It may be seen from this brief review therefore that hemorrhage into the ventricles of the brain is considered fatal, and that in the cases reported death has occurred within a relatively short time.

There are reported, however, a few cases of ventricular hemorrhage in which recovery occurred. Sanders [5] states that two of his patients recovered. He believes, moreover, that in the case of hemorrhage into

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7. Gordon, A.: Ventricular Hemorrhage: A Symptom Group, Arch. Int. Med. **17**:343 (March) 1916.

8. Allen, Alfred R.: Hemorrhage into the Ventricles. Its Relation to Convulsions and Rigidity in Apoplectiform Hemiplegia, Phil. Gen. Hosp. Rep. **7**:210, 1909.

9. Tilney, F., and Casamajor, L.: Hemorrhage into the Basal Cisterns and Ventricles of the Brain, Neurol. Bull. **1**:161 (April) 1918.

10. Laignel, Lavastine and Bloch, R.: Hemorrhagie Sous-Arachnoidienne avec inondation ventriculaire au cours d'nn nephrite subaigue, Bull. et. mém. Soc. anat. de Paris **35**:967, 1910.

11. Looten, M.: Hemorrhagie Ventriculaire Echo méd. du Nord. Lille **10**:22, 1906.

12. Dupré, E., and Sebilleau: Hemorrhagie Cerebrale. Inondation Ventriculaire, Rev. Med. **11**:341, 1903.

13. Froin, G., and Laederich: Hemorrhagie Cerebrale avec Inondation Ventriculaire et Sous-Arachnoidienne, Gaz. d. Hôp. **78**:208, 1905.

14. Abeille: Hemorrhagie et ramollissement de la calotte de la protuberance avec inondation quatrieme ventricle, Marseille méd. **41**:148, 1904.

15. Robert, A.: Hemorrhagie du cervelet; inondation ventriculaire, Progres méd. **9**:288, 1881.

16. Royster, H. A.: A Case of Cortical Hemorrhage with Rupture into a Lateral Ventricle, Med. Rec. **48**:821, 1895.

17. Sauvan, M.: Hemorrhagie cerebrale: inondation ventriculaire, Marseille méd. **44**:341, 1907.

18. Ludblum, S. D. W.: Distribution of Encephalic Hemorrhages, J. Nerv. & Ment. Dis. **36**:705 (Dec.) 1909.

19. Pope, E. L.: A Case of Hemorrhage into the Lateral Ventricle, Brit. M. J. **2**:1752, 1904.

the lateral ventricles, that "recovery is relatively frequent, as shown by the numerous recorded cases of fibrous or calcareous masses, the remains of old clots found about the choroid plexuses of these spaces, as also by two of my cases where actual and undoubted hemorrhagic remains were found. In such cases the extravasation has doubtless been small." Lannin [20] reports a case of a girl, aged 10, in whom a decompression operation was performed, following a blow to the head. Puncture of the ventricle revealed blood, and after the operation the patient made a complete recovery, leaving the hospital after twenty-two days. The question as to whether this was a true case of ventricular hemorrhage, however, is open to doubt, because one cannot rule out the possibility of error in technic.

The following case is reported as a case of ventricular hemorrhage in which the end-result was complete recovery.

REPORT OF A CASE

History.—A white man, aged 50, married, a salesman, entered the hospital, March 30, 1924, in a stuporous and comatose condition. His father is reported to have died at the age of 60 of a hemiplegia with motor aphasia. The past history of the patient was entirely negative. He had had shortness of breath for several years, had a marked polyuria, and also frequent nycturia. He had been singularly free from all disease, and had never used tobacco, alcohol or drugs.

His wife, the informant, stated that the patient had complained of severe pain over the top of his head for five or six years. These headaches were at first infrequent and felt generally over the entire head, but later became very sharp, and severe pain was felt entirely in the vertex. At times, he vomited when he had headache, but the vomiting was never projectile. Recently, the headaches had grown worse, the pains more severe and frequent, and rest was the only thing that relieved him. Since the onset of his immediate illness about three weeks before entrance, the patient had shown a marked irritability but no change of character or loss of judgment. About three weeks before entrance into the hospital, the patient worked very hard over his income tax returns, and completed them in one week, feeling so exhausted that "he could hardly move," and complaining at the same time of severe pain in the top of his head. This pain persisted for a week, and he had to give up his work. About one week before entrance, the patient was found half out of bed, apparently unconscious. He recovered in about thirty minutes, and jumped out of bed his eyes staring, foaming at the mouth, and his throat "choked up." A physician was called and said he had a slight stroke of paralysis. He was kept in bed and complained of severe pain in the top of his head. The night before entrance into the hospital, he had gone to bed quite well, but he was found in the morning unable to talk or to move his left arm or leg.

General Examination.—The patient was short of stature, pale, and comatose on entrance. The tongue was coated, and the teeth had numerous gold caps and fillings. The apex of the heart was in the sixth space. The heart showed

20. Lannin, G. E. J.: Hemorrhage into the Lateral Ventricle; Recovery, Canad. J. M. & S. **38**:119, 1915.

a supracardiac area measuring 7.5 cm., but no enlargement of the left and right borders. The heart sounds were weak and rapid. There was a systolic murmur heard best at the second right interspace, and the aortic second was accentuated. The brachial and radials were sclerosed, and the blood pressure was: systolic, 170; diastolic, 120 in the left arm; and systolic, 190; diastolic, 120 in the right arm. There was no tracheal tug, but there was impaired resonance posteriorly over the mediastinum. The lungs were clear. The liver was palpable about two fingers below the costal margin.

Neurologic Examination.—The cranial nerves showed contracted, unequal irregular pupils, reacting to accommodation but not to light. The fundi showed congested optic nerve heads, and vessels which were indicative of an angiosclerosis. There was complete ptosis of the left eyelid, and lateral mystagmoid movements. There was a right abducens weakness. The masseter and temporal muscles on the left were weak, and there was a decided facial weakness on the left side. The motor system showed a decided weakness of the left arm and leg, with the left arm held flexed over the chest and the left leg everted. The reflexes showed bilaterally diminished knee reflexes, ankle reflexes diminished with the left greater than the right, absent abdominal and cremasteric reflexes, and bilateral Babinski and Oppenheim signs. All other reflexes were normal. Sensation was not tested. Coordination, gait and other motor tests could not be made out because of the stuporous condition of the patient.

Laboratory Examination.—Temperature on entrance was 100 F., respiration 24 and pulse 108. Blood examination on admission revealed: red cells, 4,940,000; hemoglobin, 75 per cent., and white cells, 16,800. The differential count revealed 84 per cent. polymorphonuclear cells. A twenty-four hour specimen of urine showed a specific gravity of 1,034, a faint trace of albumin and a few hyaline casts. There was no sugar. Blood chemistry examination revealed: nonprotein nitrogen, 38.7 mg.; urea nitrogen, 29.2 mg. The blood Wassermann test was negative. Lumbar puncture showed a yellow fluid at a pressure of 40 mm. of water, 12 cells, no albumin or globulin, and a colloidal gold reading of 1100010000. The spinal fluid sugar content was 0.009 per cent.; the total protein, 0.027 per cent. The cistern fluid was yellow and showed a pressure of 80 mm. of water with practically the same findings as the lumbar fluid. Blood culture showed no growth after seventy-two hours. Roentgen-ray examination of the skull revealed no signs of increased pressure. The sella was normal. Roentgen-ray examination of the chest revealed a marked increase in the width of the aorta to the left, and a slight increase in the left ventricle shadow. The roentgen-ray diagnosis was an aortitis, and the opinion of the roentgenologist was that the picture was "very suggestive of aneurysm of the transverse and descending portion" of the aorta.

Course of Illness.—The patient was in the hospital from March 30, 1924, to June 3, 1924, a period of approximately nine weeks. The immediate treatment consisted of cardiac stimulation because of the extremely weak pulse. Lumbar puncture was performed four times in five days, and cisterna puncture was performed twice on the day of entrance and on the day following. After both the cistern and the lumbar punctures, the patient was much quieter and rested more easily.

Hypertonic saline (15 per cent.) was given intravenously over a period of three weeks in doses of from 100 c.c. to 200 c.c. For the first week, this was given as often as three or four times a day, but after this the hypertonic saline was administered when there seemed to be evidence of an increasing intracranial pressure or of return of the headache.

From March 30, 1924, to April 6, 1924, the patient remained stuporous, and apparently confused and disoriented. He took liquid foods. His physical status remained as described above. His condition was critical, especially the first two or three days after entrance, and on the second night he developed typical Cheyne-Stokes respiration. After a week in this condition, however, he seemed to show some improvement. On April 6, one week after entrance, he was quite bright and rational, and seemed to have emerged from his stuporous condition. The ptosis of his left eyelid was less; he could now open his eye a little, but the left facial palsy and left hemiplegia remained. He continued to improve daily. His ptosis gradually cleared. His speech was at first thick, but on April 9 it was noted that the Babinski reflex on the right had disappeared, but that the one on the left persisted. He could now move his left hand to his mouth, and could move his leg a little. By April 15, the patient could walk; he was ataxic and dragged his leg, but needed no support. His speech was clearer, and drooling much less than on entrance. The ptosis had completely disappeared, the left side of the face was weak, but it was definitely better than on entrance, and the drooling was much less marked. On April 28, the Babinski sign on the left had disappeared, and the ankle and knee reflexes were now active and apparently normal. From April 28 to the date of his discharge, June 3, 1924, the patient made steady and continuous improvement. He continued to gain strength in his left arm and leg, and his speech and drooling gradually improved. On May 19, there was still some weakness of the left side of the face; the movements of the arms and legs were good; the reflexes were normal, and there was no Babinski sign on either side. The patient improved until on discharge, June 3, 1924, he had no weakness of the left arm or leg, and was drooling very little. The left side of his face was still somewhat weak.

From March 30 to April 6, 1924, the temperature varied from 100 to 103 F. On April 7, the temperature returned to normal and remained there until discharge. The number of leukocytes varied from 11,400 to 23,000 from March 30 to May 2. On May 6, the number of leukocytes was normal, and they varied from 7,200 to 8,800 from May 6 until the patient was discharged. Repeated examination of the urine revealed a specific gravity from 1.015 to 1.030, a faint trace of albumin and hyaline casts during most of the examinations. On discharge, the urine still showed a faint trace of albumin and occasional hyaline casts. The blood pressure on discharge was: systolic, 156; diastolic, 96. The renal function shortly before discharge was 85 per cent. The fundi showed the same findings on discharge as on entrance.

DISCUSSION

The diagnoses considered on entrance were cerebral hemorrhage, brain tumor with hemorrhage, epidemic encephalitis and central nervous system syphilis. The widespread signs found in this case could be produced only by an extensive and therefore fatal hemorrhage. The absence of the ordinary premonitory signs of increased intracranial pressure and the absence of choked disks seemed to rule out a brain tumor. Epidemic encephalitis was ruled out by the absence of any definite history or signs which might prove the existence of this infection, and by the complete recovery of the patient. Neurosyphilis was excluded by the negative serology. The diagnosis of ventricular hemor-

rhage seemed justified on the basis of the widespread neurologic signs, the repeatedly bloody spinal fluids, and the rapid recovery which would seem to point to a hemorrhage into an area where absorption could readily take place. One finds the basis for the hemorrhage in the presence of cardiorenal disease with an increased blood pressure. Sanders [5] mentions as one of the causes of ventricular hemorrhage, aneurysms encroaching on or existing within the ventricles, and it may be that this was the cause of this case, particularly since there seemed to be evidence of aneurysm elsewhere in the body. The fact that the spinal fluid was not frankly bloody does not exclude the possibility of a ventricular hemorrhage for Dupré [12] and Froin [13] report cases of ventricular hemorrhage in which blood was found in the spinal fluid only on centrifugalization. In the former case, the fluid was clear, and in the latter, it was yellow. Both cases were confirmed by necropsy.

No general conclusions can be drawn as to the treatment of ventricular hemorrhage from this one case. In this instance, however, frequent drainage of the spinal fluid by cistern and lumbar punctures was effective in producing beneficial results. The use of hypertonic saline also was of great value in decreasing the spinal fluid pressure, and thus decreasing the intracranial pressure. It may be that the more frequent use of repeated punctures in ventricular hemorrhage may be of value in other cases.

How are we to explain the temperature and leukocytosis? Tilney and Casamajor [9] report a slight rise in temperature of 100 to 101 F. following hemorrhage into the basal cisterns and ventricles. Musser [21] reports a leukocytosis following hemorrhage, and Drinker [21] holds that "there are few biological reactions which may be so fully relied on as the leukocytosis following hemorrhage."

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21. Musser, J. H., Jr.: The Leucocytes After Hemorrhage, Am. J. M. Sc. **162**:40 (July) 1921.

News and Comment

ASSOCIATION FOR RESEARCH IN NERVOUS AND MENTAL DISEASES

The meeting of the Association will be held, December 29 and 30, 1924, at the Hotel Commodore, New York City. The tentative program follows:

Historical Résumé of the Knowledge of the Human Cerebrospinal Fluid. *Dr. Walter Timme.*

Embryogenesis of the Human Cerebrospinal Fluid; Its Source, Circulatory Pathways, Destination, Together with Its Biological Significance in Relation to the Blood Vessels and Lymphatics. *Dr. Walter Hughson.*

The Normal Human Cerebrospinal Fluid; Its Cytology, Biologic and Physical Properties, Together with the General Principles Governing Its Deviations from the Normal; Its Differences in Various Age Epochs. *Dr. C. Burns Craig.*

The Human Cerebrospinal Fluid; Its Chemical and Physical Properties under Normal and Pathologic Conditions.

(*a*) Examination of the Fluid from Different Loci; Cerebral and Spinal. (Reader not yet assigned.) Massachusetts General Hospital and Psychopathic Hospital.

(*b*) Quantitative Chlorids. *Dr. Frank Fremont-Smith.*

(*c*) Comparison of Three Colloidal Tests, Gold Chlorid, Benzoin and Mastic Tests. *Jessie R. Cockrill.*

(*d*) Quantitative Protein in Different Conditions. *Dr. James B. Ayer.*

(*e*) Quantitative Sugar, Normal and Pathologic. Correlation with Blood Sugar. *Dr. Frank Fremont-Smith.*

(*f*) Electrical Conductivity of the Spinal Fluid in Health and Disease. *Dr. John L. Eckel.*

Human Cerebrospinal Fluid Pressure Studies.

(*a*) Pressure Studies, Normal and Pathologic. *Dr. James B. Ayer.*

(*b*) Manometric Studies of the Human Cerebrospinal Fluid in the Diagnosis of Spinal Cord Neoplasms. *Dr. Byron P. Stookey.*

(*c*) Experimental Studies in Increased Intracranial and Subarachnoid Pressure. *Dr. Hubert S. Howe.*

(*d*) Experimental Pressure Studies with Different Concentrations of Salts. *Dr. W. L. Aycock.*

(*e*) The Increase of Cerebrospinal Fluid Pressure as a Problem on the Operating Table. *Dr. Charles H. Frazier.*

(*f*) Combined Ventricular and Lumbar Punctures for the Localization of Brain Tumor. *Dr. Frank Fremont-Smith* and *Dr. John S. Hodgson.*

Ventriculography, Direct and Indirect. *Dr. F. C. Grant.*

The Value of Lipiodol in the Localization of Spinal Lesions. *Dr. W. J. Mixter.*

The Bacteriology of the Pathologic Human Cerebrospinal Fluid and Its Modifications in all Respects Due to Bacteria, Protozoa and Other Micro-Organisms. *Dr. John A. Kolmer.*

The Effect of Central Nerve Changes on the Subarachnoid Space, Choroid Plexus and Cerebrospinal Fluid. *Dr. George B. Hassin.*

The Reaction of the Human Cerebrospinal Fluid in Infectious Inflammatory Conditions in Cranial and Intracranial Disease. *Dr. Israel Strauss.*

The Human Cerebrospinal Fluid in the Extraneural Acute Infectious Diseases. Statistical tabulations by

Dr. Oscar M. Schloss
Dr. Josephine B. Neal
Dr. Stafford McLean
Dr. Joseph C. Regan

The Human Cerebrospinal Fluid in General System and Metabolic Diseases, as in Nephritis, Diabetes, etc. *Dr. B. J. Alpers.*

The Human Cerebrospinal Fluid in Chronic Infectious, System and Diffuse Degenerative Diseases Involving the Nervous System. *Dr. C. A. Patten.*

The Spinal Fluid in Syphilis of the Nervous System. *Dr. Harry C. Solomon.*

The Spinal Fluid in the Non-Syphilitic Psychoses. *Dr. Charles Macfie Campbell* and *Dr. George H. Kirby.*

The Spinal Fluid in Brain Tumors. *Dr. Spurling.*

Carbon Monoxid Poisoning. Experimental and Clinical. *Dr. Harry S. Forbes.*

The Cerebrospinal Fluid in Lead Poisoning. *Dr. Carl V. Weller.*

The Intravertebral Treatment of Diseases of the Central Nervous System Exclusive of the Intraspinal Treatment of Cerebrospinal Syphilis. *Dr. Lewis J. Pollock.*

The Intraspinal Treatment of Cerebrospinal Syphilis. *Dr. John A. Fordyce.*

Abstracts from Current Literature

HEMOHYDRAULIC ENCEPHALO-MEDULLARY PHENOMENA. STUDIES ON CEREBRO-SPINAL CONCUSSION (COMMOTIO). FRANCESCO PEDRAZZINI, J. f. Psychol. u. Neurol. **30**:129, 1924.

The term "commotio cerebrospinalis" is used by Pedrazzini to designate those manifestations on the part of the central neuraxis that follow elastic deformities of the craniospinal bones in consequence of the static or dynamic transmission of a blow. The impact of a blow or of a compression of the cranial vault which does not produce a fracture gives rise to an elastic deformity of the skull. This is due to the inherent elasticity of the osseous structure of the skull bones and to their proper adjustment. The pathogenetic elements of concussion consist, on the one hand, of the intensity, rapidity, extent, direction and location of the blow, and on the other, of the physical properties of the resisting solid body, the skull, through its effects on the hemo-hydraulic apparatus, and in cases with dynamic transmission of the blood, on the cerebrospinal mass itself. When the patient does not succumb to the trauma, there appear certain biologic manifestations that are to be regarded merely as phenomena of reaction; they are the sequelae of cerebral concussion, and, as such, have really nothing to do with the pathogenesis of the latter. The mechanics of concussion depend largely on the fact that the deformity of the osseous structure is transmitted to its underlying dural sac and to the cavities which contain the cerebrospinal fluid. But as the latter (the cerebrospinal fluid) is not compressible, it acts as a medium which transmits most directly the blow to the fluid containing chambers; this transmitting energy, however, is in part minimized or overcome by the succeeeding, elastic deformity of the dural sac and of the fluid containing chambers.

In experimenting on dogs, Pedrazzini found that when he introduced a cannula into the subarachnoid space of an animal while at rest, the manometer registered a rise in the pressure of the cerebrospinal fluid every time that pressure was exerted on the cranial vault, provided the pressure was sufficiently intense to diminish the intracranial capacity; this rise in pressure lasted as long as the deformity of the skull, induced by external pressure, was kept up, and receded as soon as the cranial vault had regained its original capacity. From this he concludes that the noncompressible cerebrospinal fluid within the cannula expands and rises as high as the volume of the induced deformity of the skull permits it to rise.

In cases in which the impact is not a sudden one, and the deformity of the skull (if it does not lead to fracture) does not last sufficiently long to transmit the effects of pressure to the cerebrospinal fluid, the brain at once begins to feel the entire effect of the compression. This is in accordance with Pascal's law of a static fluid. Should, at this time, however, the balancing between the spinal fluid and the ventricular fluid pressures come into play, the rise in pressure may be reduced to very low figures, especially when the resistance of the elastic spinal sac, and that to the flow of the fluid through the ventricular foramina, become diminished. In this case the brain hardly feels the effect of the impact, and, even in cases in which the impact is of considerable intensity, or while carrying an unusually heavy weight on the head, no pathologic effects will follow.

As a result of these physiologic manifestations, one would expect that the skull deformity would at first seek to recover its balance at the expense of the spinal column, because it is much easier for the spinal dura to release its tension than for the spinal fluid from the arachnoid spaces to enter the ventricles. Should, however, for pathologic reasons the spinal dura be thickened and more rigid, so that it becomes more difficult for it to relax its tension than for the spinal fluid to penetrate into the ventricles, the balancing of the post-traumatic elastic cranial deformity would take place in the ventricles themselves. This equalization of tension may, therefore, occur in the spinal dura as well as in the ventricles. In any case, the rule is that an excess in the fluid volume due to a diminished capacity of the elastic fluid chamber seeks to equalize itself in another portion of the latter, and this is accomplished by a corresponding increase in the capacity or by an increased accumulation of fluid induced by a transmission of the impact through a pressure wave.

As long as all other conditions within the system are in harmony with these phenomena, the neuraxis experiences no ill effects. Should, however, their intensity not be commensurate with the delicate structure of the tissues, there result pathologic sequelae that have their point of origin within the elastic deformity of the skull, with the ill effects of the latter centering themselves on the elastic dural sac and the cerebrospinal fluid within the subarachnoid spaces. In cases of fracture, the general cerebral disturbances as well as the local disturbances situated remotely from the area or areas on which the impact has exerted its greatest force, are also to be attributed to the elastic deformities of the bony wall, because the latter had occurred prior to the fracture. But in cases of skull fracture which leads to a perforation of the fluid-containing arachnoid chamber, there occurs neither an "elastic" nor an "impact" effect. In the temporal region, where the degree of resistance is less than in the cranial vault, impacts and blows are followed more readily by fracture or by separation of the sutures. It is for the same reason that in these cases local brain lesions are found more frequently than indirect lesions, due to the transmission of the impact or blow through the cerebrospinal fluid. In this connection, it must also be borne in mind that as the pressure effects from the elastic skull deformities are, according to Pascal's law, uniformly transmitted, the leptomeninges of the subarachnoid spaces, as well as those covering the cerebral cortex, are equally affected.

During cerebral concussion there occurs a rupture of some of the delicate vessels of the pial network or of the cortex, so that lumbar puncture frequently yields a blood-tinged or bloody fluid rich in erythrocytes. Necropsy shows meningeal as well as cortical ecchymoses. In cases in which a long period of time has elapsed between the trauma and death, the delicate cerebral coverings contain whitish spots—evidences of irritation, and connective tissue hyperplasia of these membranes. Coma appearing immediately after the trauma—evidence of a disturbance in function of the cortical cells—is so frequent that it is commonly mistaken as the cause rather than the effect of the injury. Unconsciousness and coma may be slight and transitory in nature; they may be entirely absent, or they may develop late, and remain severe and lasting sequelae.

Following cranial trauma, there occasionally occur changes in the fundi oculorum, such as a filling up, tortuosity, dilatation and varicosity of the retinal veins and papilledema. These changes are not unlike those observed in cases of slowly increasing intracranial pressure due to meningitis, cerebral tumor and hydrocephalus. Sudden increase of intracranial pressure gives rise to a

sudden and violent closure of the cavernous sinus, a sudden overfilling of the central retinal vein and a dilatation of the veins of the papilla—the latter, owing to the short duration of the obstruction to the outflow, often regain their original size so that no traces are left of the temporary circulatory disturbances. But when the venous distention is marked so that the elasticity of the walls of the vessels is diminished or their nutrition disturbed, these circulatory disturbances are of much longer duration; it may take a long time for them to regain their normalcy; they may remain stationary or they may progress to edema and elevation of the disk. These changes are due to mechanical factors as well as to biologic reaction phenomena that take place within the dural sac, in consequence of which the increased intracranial pressure remains stationary or keeps on increasing.

Owing to the physical relationship existing between the cerebrospinal fluid, the arterioles and the encephalomedullary capillaries and veins, a sudden change of equilibrium in the pressure of the cerebrospinal fluid leads to a disturbance of the entire cerebrospinal circulation. This accounts for the hyperemia, small hemorrhages and nutritional disturbances of the small blood vessels and the cellular elements themselves as well as for the changes in the phenomena of filtration and osmosis, which give rise to the presence of erythrocytes and abnormal products of metabolism in the cerebrospinal fluid, hyperalbuminosis, increase in the quantity as well as in the pressure of the cerebrospinal fluid—manifestations that are actually encountered in cases of cerebral concussion. Lumbar puncture, which serves to diminish the amount and pressure of the cerebrospinal fluid, i. e., the hypertension of the elastic dural sac (without abolishing it entirely) to promote the cerebral circulation without changing its physiologic type, to drain the ventricles when they are overfilled with fluid, as well as to remove toxic substances from the cerebral circulation, is not less advantageous for the purpose of combating the manifestations of cerebral concussion than for the relief of choked disk. However, as the mechanical factor represents only the "primum movens," and the hypertension is maintained solely by the effects of the latter (circulatory disturbances and disturbances in cellular life), Cantonnet has observed that the first puncture is usually followed by slight improvement, whereas a second puncture yields much better results. Several times a third puncture was necessary. The duration of the headache runs parallel with the papilledema.

In his experiments on dogs, the author frequently found that puncturing the dura and arachnoid with a needle was sufficient to increase the elasticity of the dural sac and to diminish the pressure as well as the fluctuation level of the cerebrospinal fluid. But occasionally the pressure and fluctuations of the fluid as well as the tension of the dural sac would not be affected even after larger incisions or after ablation of a considerable amount of dura and the other cerebral coverings through a large sized trephine hole. Physically, this can only be explained by the fact that when the arachnoid sac is practically closed by adhesions and a matting together of the arachnoid and pia around the artificially produced opening, or by the wedging in of brain substance (herniation), the dural sac becomes actually totally shut off. This explanation, the author believes, is also applicable to the mechanism of papilledema in cases of cerebral injury.

A dynamic transmission of the impact occurs in cases in which, owing to the lack of time, Pascal's law fails to operate. If the blow be sudden and the deformity of the skull remain within its limits of elasticity, the subjacent cerebrospinal fluid has no time to rebound against the entire sac, and acts as a

solid body transmitting the blow directly to the cerebral mass contained within the sac. In this connection, it is interesting to see how the force transmitted from the site of the impact is lost and what the effects of such loss of force are. A study of the encephalitic changes following the failure of this force serves this purpose best, because the site or sites of these changes indicate the area or areas in which most of the transmitting force or energy is lost. As, on account of the suddenness of the blow, the cerebrospinal fluid has not sufficient time to be displaced, there occurs no equalization between the elastic deformity of the skull and the stretching of the dural sac and the fluid accumulated in the ventricles. An impact sufficiently intense to lead to a cranial deformity must of necessity reduce the volume of the cranial contents. But here it must be borne in mind that the latter consist chiefly of the noncompressible cerebrospinal fluid and the yielding and compressible nerve substance. As a matter of fact, the compressibility of the brain is, under physiologic conditions, much greater because the ventricles are not completely filled with fluid, so that there are formed cavitations within the brain into which the nerve substance can readily sink. Thus during a sudden blow, associated with inactivity of the cerebrospinal fluid, elastic deformity of the skull becomes possible only through the compressibility of the brain itself. The fluid layer underneath the site of the blow transmits the latter, like a solid body, to the cerebral cortex, which, because of its compressibility, experiences a depression similar to that seen in the elastic deformity of the cranial bones. At necropsy, one frequently finds under the surface of the part of the skull affected by the blow, even when the bone itself remains intact, lesions such as ecchymoses of the meninges and of the cerebral cortex. When the entire force or energy of the blow has spent itself in loco, other areas are free from lesions, but when some of the energy has not been completely exhausted at the site of impact, some of it is transmitted to other parts of the brain with resulting lesions also in these areas. Furthermore, it may also happen, though rarely, that the entire force of the impact or the greatest part of it is not exhausted at its site but is transmitted directly to the entire brain; in such cases the manifestations are those of a blow from within. This readily explains why a trauma to the convexity of the skull, through a dynamic transmission of the blow, although the bone remains intact, gives rise to ecchymoses and cortical lesions at points contre coup or remote from the site of impact. It also explains the mechanism of the appearance of injuries and of the immediate as well as of the late hemorrhages in the deeper parts of the brain (central ganglia, pons and medulla).

The direction of the blow also influences the site of the concussion. A blow directed so that the brain is displaced from above downward gives rise to a concussion of the deeper or basal parts.

Falling on the buttocks or lower limbs, even though the spinal column remains intact, produces deformities with changes in form and diameter in various sections of the spinal canal. These are due to the resulting stretching and elastic reaction of the vertebral column, to its natural curves, to the suppleness of the limbs and to the elasticity of the column itself. But when, in spite of the presence of the fatty tissue and the arterial plexus between the dura and spinal canal, these deformities lead to a diminution in the capacity of the dural sac, there occurs a pressure wave in the cerebrospinal fluid against those parts in which the pressure is lowermost. The length of the vertebral column and the numerous anastomoses between the intraspinal and extraspinal arterial plexuses readily equalize this pressure. The fact that the spinal pia is more of a protective membrane than the cerebral pia accounts for the mildness

of symptoms and for the lesser frequency of spinal concussion as compared to cerebral concussion. Elastic deformities of the spinal column occurring during jumping are therefore regularly well borne, although even here one may occasionally encounter hemorrhages within the cord, myelitis, tabes, amyotrophic lateral sclerosis, anterior poliomyelitis and syringomyelia of traumatic origin. (The author probably employs these terms to designate pathologic lesions in the cord that assume the patterns of the diseases mentioned above—*Reviewer.*) Falls on the buttocks or feet more frequently give rise to conus lesions. The latter follow falls on the buttocks more often than on the feet, because the suppleness of the joints of the lower limbs has a great tendency to minimize the effects of the impact. The usual bending of the trunk forward rather than backward is probably due to the direction of the fall. Of significance is also the fact that the conus is surrounded by the spinal fluid as well as by the elastic dural sac which encloses the fluid and cord as if in a fibrous case. The conus is also held in position by the spinal roots and the cauda equina fibers, whereas the dural sac is padded out externally by perimeningeal fatty tissue and by the intraspinal epidural arterial plexus. Should the trauma produce a severe stretching of the spinal column, it would seem that the spinal roots and the cauda equina fibers would be the first to feel the ill effects of the trauma. The spinal fluid between the termination of the cord and the end of the spinal canal does not serve so much to diminish the pressure waves coming from the cranial cavity as to protect the cōnus against the waves which compress directly the conus while jumping or falling on the buttocks or feet. This is the case as long as the transmission of the effects of compression is, according to Pascal's law, well balanced and equalized at the proper time. Should, however, the blow or impact be a severe and sudden one, as in cases in which the buttocks alone are involved, then the spinal fluid which has received the impact transmits it to the conus—the fluid has overcome the resistance of the impact itself and that of the cauda equina fibers—so that the conus alone is injured.

Involvement of the neuraxis from explosions of large firearms at certain distances is also due to the elastic deformities of the elastic dural sac. The World War has brought out two principal facts: 1. Explosions from large guns situated at a distance can produce the same manifestations of concussion of the neuraxis as those following any trauma to the skull or spinal column. 2. In addition to the nerve injuries, such explosions also produce pathologic changes in the viscera of the large splanchnic cavities—bronchial bleeding, hematuria, rupture of the cardiac valves and disturbances of the peritoneum—phenomena that are never observed in trauma involving the skull and spinal column alone. This association of visceral and nerve lesions is helpful in the study of the mechanism of such lesions.

The pressure of air from an explosion of a large sized shell in the second zone of explosion affects the superficial surface of the body, causing compression of the soft and flexible parts: the anterior abdominal wall, the ribs and the soft parts of the neck. The impact is also felt, although to a somewhat lesser degree, by the more compact and resistant tissues, such as the musculature of the limbs, and to a still lesser degree by the rigid parts of the body, such as the bones of the skull and the vertebral column. Forcible compression of the trunk and abdominal walls gives rise to increased pressure in the corresponding cavities, with a consequent sudden reduction in the size of the large venous branches of the trunk and abdomen, of the iliac veins, the azygos veins, the venae cavae and of the auricles themselves. The pressure remains unchanged

in the vertebral canal and in the brain because of the rigidity of the skull. The blood then flows from the venous branches of the larger splanchnic cavities with increased pressure into the intraspinal venous plexus; as a result of the increased pressure in this plexus there occurs a sudden dilatation of it, which, in turn, gives rise to a sudden increase in the volume content of the perimeningeal space with a gradual contraction of the medullary dural sac as well as to a marked increased pressure wave in the cerebrospinal fluid. At the same time, the force of the explosion causes the air to compress the structures of the neck, retarding the outflow of blood from the jugulars and the intracranial vessels. These phenomena, therefore, give rise to a summation of cranial and spinal effects, with positive signs. An equalization or balancing of these effects can occur only as a result of the compressibility of the nerve substance and of the accumulated cerebrospinal fluid in the ventricles. Immediately after the explosion, the displaced air returns to the "explosion cone," giving rise to manifestations in an opposite direction. The encephalo-medullary lack of balance in the vessels, the fluctuations in pressure as well as the displacements of the cerebrospinal fluid of the first or positive phase, become through the summation of effects more potent than the effects of the second or negative phase.

From this entire study the author concludes that all manifestations of concussion of the central nervous system depend on the elastic deformities of the elastic dural sac, when the latter is held in tension.

KESCHNER, New York.

THE PATHOLOGY OF THE PARKINSONIAN SYNDROME FOLLOWING ENCEPHALITIS LETHARGICA, WITH A NOTE ON THE OCCURRENCE OF CALCIFICATION IN THIS DISEASE. DOUGLAS MCALPINE, Brain **46**:255 (Oct.) 1923.

The author begins with a review of the literature on the pathology of paralysis agitans after epidemic encephalitis. Marinesco reports a case in which there were intense lesions in the locus niger, with cytolysis, accumulation of pigment, neuroglial infiltration, a loss of nerve cells and perivascular infiltration. The globus pallidus showed changes, but here they were less intense than in the locus niger. McKinley reports a case with comparatively insignificant findings in the globus pallidus, putamen and caudate nucleus, and with intense changes in the substantia nigra consisting in a moderate amount of chromatolysis, marked reduction in the number of cells, decrease in the number of the axis cylinders, increase in glia and perivascular infiltration. Foix reports a case in which the lesions were greatest in the substantia nigra. Francais and L'Hermitte report a case with marked pathologic findings in the substantia nigra, as detailed above, and with marked changes also in the pallidal system. L'Hermitte concludes that the symptoms in this case were due to involvement of the pallidal system.

Mlle. G. Levy discussed the pathologic findings in four cases of paralysis agitans following epidemic encephalitis. She lays stress on perivascular disintegration in the basal ganglia. She describes changes in the ansa lenticularis, internal capsule, corpus Luysii, red nucleus and locus niger. She concludes from the widespread nature of the process that no conclusions can be drawn as to the pathogenesis of paralysis agitans.

Goldstein reports a case in which there were marked changes in the substantia nigra. Tretiakoff and Bremer report a case in which the most marked

changes occurred in the mesencephalon, and particularly in the locus niger, in which the number of cells was much reduced. Inflammatory changes consisting of increase in lymphocytes, congestion of vessels, and moderate neuroglial growth were present. The globus pallidus was free from inflammatory changes.

D'Antona and Vegni and then again Vegni report cases with marked degenerative changes in the substantia nigra. Gamma and Omodei-Zorni report two cases with intense changes in the substantia nigra and hypothalamus; both their cases were cases of encephalitis of the choreiform type, which later developed into a parkinsonian syndrome.

Urechia reports a case of postencephalitic parkinsonian syndrome with intense inflammatory changes in the basal ganglia, especially in the globus pallidus, corpus Luysii and substantia nigra.

The author reports two cases of postencephalitic parkinsonian syndrome. In both cases, subacute inflammatory changes were found, most marked in the mesencephalon. In Case 2, the changes were slighter than in Case 1, and were more marked in the basal ganglia and subthalamic region than in the mid-brain. In Case 1, there was a marked diminution of the cells in the substantia nigra; in Case 2, the cells showed no reduction in number. The author calls attention to the mode of death in both cases, viz., sudden pyrexia and tachypnea, which were progressive. In both cases, calcareous deposits were observed in the walls of the vessels in the anterior part of the globus pallidus and in this region only.

Two schools of thought exist on the pathogenesis of paralysis agitans. The one represented by the Vogt's, L'Hermitte, Ramsay Hunt and others, considers that the causative lesion in idiopathic paralysis agitans lies in the globus pallidus. The other, led by Tretiakoff, who is supported by Pierre Marie, thinks the disease is due to lesions in the substantia nigra. The former bases his opinion on a study of twenty-nine cases representing all sorts of organic disease of the nervous system. Of these, twenty cases showed no changes in the substantia nigra, while nine cases of idiopathic paralysis agitans showed degenerative changes there.

The author examined the substantia nigra in cases of myasthenia gravis, cerebral syphilis, subacute combined degeneration of the cord, primary dementia, myotonia atrophica and recurrent cerebral hemorrhage. In none of these did he find changes in the substantia nigra.

Three cases of idiopathic paralysis agitans showed a normal substantia nigra. Three cases of epidemic encephalitis, however, showed marked changes in the substantia nigra. McAlpine therefore concludes that "in idiopathic paralysis agitans a lesion of the globus pallidus, or possibly of the subthalamic structures with which it is connected, is the usual pathological basis of the disease; whereas post-encephalitic Parkinsonianism is usually due to degeneration of the substantia nigra."

In 1918, Buzzard and Greenfield first called attention to the occurrence of calcification in epidemic encephalitis. They described calcification of a few vessels of the cortex, and more particularly of the vessels of the globus pallidus in one case. Dürch, in 1921, found calcification in twelve of fifteen fatal cases of epidemic encephalitis. The calcification of vessels occurred most frequently in the corpus striatum, but also were found in the dentate nucleus, hippocampus, cornu ammoni, hemispheres and optic thalamus. The author examined the brains in six fatal cases of epidemic encephalitis. In all six cases, calcification in varying forms was found. In all the cases except one, the process was

limited to the anterior part of the globus pallidus. Controls were made on brains from cases of trauma, cerebral tumor, subacute combined degeneration of the cord, transverse lesion of the cord in the cervical region, Huntington's chorea and three cases of idiopathic paralysis agitans.

Well marked calcification of the vessels in the anterior part of the globus pallidus was found in the case of Huntington's chorea. Similar changes were found in other brains.

The histologic changes found in the vessels are described in detail. These may be summarized as follows: (1) a hyaline degeneration of the media; (2) a fatty degeneration of the vessel walls; (3) the occurrence of calcium deposits between the adventitia and media.

Calcification in the vessels of the brain have also been described in arteriosclerosis. Dürch has found calcification of the cerebral vessels in the brain of an idiot. Dürch and Weingarten have both observed calcification of the vessels in the corpus striatum in cases of malignant malaria. Weiman has reported a case of long standing dementia in which calcification was widespread.

ALPERS, Philadelphia.

PSYCHONEUROTIC DISTURBANCES IN HYPERTHYROIDISM. R. F. WEISS, Ztschr. f. klin. Med. **97**:366 (Sept. 20) 1923.

Renewed interest has been shown of late in the relationship between psychoneurotic disturbances and internal secretion. The thyroid is supposed to exert considerable influence on psychic life, especially of the affective type. Thus, Rothschild and Levi have considered the thyroid the "glande de l'emotion."

Recently, Ritterhaus has attempted to attribute the phases of manic-depressive insanity to changes in thyroid function. Nervous and psychic disturbances in exophthalmic goiter are well known, but their exact significance is not clear.

Two opposing views are held in regard to the psychoneurotic disturbances associated with hyperthyroidism. The first is that the psychic manifestations, as all other manifestations, are the result of thyreotoxicosis. On the other hand, some wish to consider the nervous and mental phenomena as entirely, or at least in part, unrelated to the thyroid, and not primarily related to the disease process. It has long been recognized that exophthalmic goiter is especially prone to occur in nervous persons, who often have a nervous heredity. Charcot and Féré included exophthalmic goiter in their neuropathic groups. Bauer has considered this entire group under the head of thyreotoxic constitution. Kraus stated that the thyroid neurosis is not entirely the result of thyroid, but that a constitutional element is also a factor. Chvostek, in his monograph, asserts that exophthalmic goiter is an exquisite degenerative process and that numerous nervous phenomena may be present without being definitely related to hyperfunction. Thus some of the most severe cases of nervous changes are present in mild thyroid cases.

Rather characteristic of the nervousness in exophthalmic goiter is the patient's complaint of nervousness, over which he is extremely unhappy. This is contrary to the neuropathic person.

The author reports seven cases in which definite signs of marked hyperthyroidism existed over a considerable period of time. All of the patients made a good physical recovery, two following operation, one following radium therapy and four as the result of medical care. The psychoneurotic symptoms,

such as anxiety attacks, emotional instability, fears and obsessions, continued, and the patients had to be classified as psychopathic personalities or constitutionally minderwertig.

From these observations, Nissen feels that it may be stated that the psychoneurotic complex is essentially independent of the hyperthyroidism and that pathologically there is no relation between the two conditions. It is possible for either of these two components to vary considerably in their severity without having any influence on the other component. It is also true that the same psychoneurotic symptom may occur in persons who show no evidence of thyroid disturbance.

Hyperthyroidism tends to increase the sensibility of the nervous system. Due to this increased sensibility, neurotic symptoms may readily arise. It is also true that with the increase of neurotic symptoms, such as anxiety and fear, thyroid symptoms may increase. Some authorities recognize a psychosis of exophthalmic goiter and, as stated previously, have looked on maniacal reactions as the result of thyroid activity.

In attempting to solve the relationship between the psychic and hormonal influences in such a problem, one first turns to the vegetative nervous system. Bower has placed the thyroid in this vegetative nervous system complex. It acts as a multiplier, which may be stimulated either by the vegetative nervous system or conversely by thyroid overactivity. This is noted clinically, as psychoneurotic persons show that either the entire or a portion of the vegetative nervous system is overactive, as demonstrated by marked perspiration, tachycardia and dilated pupils. It would seem that both conditions are related or dependent on the vegetative nervous system for their existence, rather than on one another. Close analysis shows these disease pictures to resolve themselves into two components, which may be interrelated although there is no common pathologic basis.

The psychic component, whose seat is in the cerebrum, and the hormonal component, functioning through the hormonal system, acting in a constitutionally oversensitive person, are bound together through the vegetative nervous system. Both components may be observed during the disease process. For a time, the neurotic symptoms are in the foreground; again there may be an exacerbation of hyperthyroidism. The basis for these components is a constitutional defect involving both the psychoneurotic foundation and the endocrine system. Nissen believes that the terminology in regard to this syndrome has been much confused. All psychoneurotic persons do not have hyperthyroidism and all persons with hyperthyroidism need not show marked psychoneurotic symptoms. The term thyroid neurosis should be used cautiously, as the syndrome represents a neurotic thyreotoxic syndrome, and is based on a constitutional factor.

The therapy must be directed against both the thyroid and the neurotic symptoms. Complete restitution is hardly to be expected. In discussing the medical treatment, Nissen takes up both the psychoneurotic and the hyperthyroid symptoms. Regarding operative procedures, he is not enthusiastic, especially in the group in which the neurotic symptoms predominate. He warns against such a measure unless the indications for operation are definite.

In summarizing, Weiss points out that there is a definite disease picture, which is characterized by numerous psychoneurotic symptoms which are constantly associated with an increase of thyroid function. Analysis of this disease group shows no underlying basic pathology, but the two components of the disease are dependent on the vegetative nervous system. The disease picture,

a neurotic thyreotoxic syndrome, is an indication of an underlying constitutional defect. All degrees of severity may occur, but usually the psychoneurotic symptoms are the most pronounced. The therapy must be directed toward both components, but mainly against the psychoneurotic symptoms.

Unfortunately, in the author's works there have been no thorough metabolic studies reported, which necessarily throws some doubt on his conclusions.

MOERSCH, Rochester, Minn.

THOMSEN'S DISEASE (MYOTONIA CONGENITA). KARL NISSEN, Ztschr. f. klin. Med. **97**:58 (Feb.) 1923.

Historical: Nissen reviews fully the subject of myotonia congenita. Thomsen, who was his grand-uncle, believed that the condition was described as early as 1832 by Charles Bell. Following the work of Thomsen, several cases were reported, but the disease was not fully described until 1886, when Erb did so in his classical monogram "Die Thomsensche Krankheit." Erb divided the cases into (*a*) the pure or typical cases, (*b*) the doubtful cases and (*c*) the symptomatic cases. Erb's pathologic findings, as also his myotonic reactions, are now well known and described in all textbooks on neurology.

Nissen is of the opinion that many of the cases reported in which the myotonia occurs only in the cold, as described by Eulenburg, should be termed "paramyotonia hereditaria" and should not be confused with myotonia congenita. He believes that "myotonia intermittens congenita" and "myotonia acquisita" are probably true myotonias which may have been latent or of a mild character, and which may have existed without the apparent knowledge of the individual. This idea is in accordance with the views of Curschmann and Pelz. The author considers myotonia atrophica (Hoffmann) as a disease *sui generis,* not a true myopathy, but a distinct atrophic diathesis.

Symptomatology: The characteristic symptom of Thomsen's disease is the intention rigidity, or muscular contracture of all striated muscles, especially of the extremities, but also of the face, tongue and eye muscles. The associated myotonic electrical reactions are noteworthy. Nissen has been able to trace the disease in his own family through seven generations. In infants, it is difficult to make a diagnosis, as the movements are so nonphysiologic. The first symptoms are noted in the facial musculature, especially the difficulty in looking upward. By the time the child begins to walk, the muscular rigidity may be observed. As the patient develops, more and more muscular contractions may be observed.

From a psychic standpoint, Thomsen was of the opinion that senile changes occurred especially frequently. The author doubts this, and was unable to substantiate such a finding. As far as he was able to determine, psychic manifestations seemed rather rare in this disease. In determining the hereditary factors, Nissen traced the disease through seven generations, including 219 persons. In accord with Rudin, he found that the affection followed the mendelian law. Five chief factors presented themselves: (1) direct heredity, (2) transmission only through dominants, (3) once free, always free, (4) the result of one-half dominance and one-half unaffected if a dominant marries a healthy person, (5) the majority of members in large families affected.

According to Nissen's observations, if two recessives marry, no dominance will appear, but the condition will remain recessive. From this heredity study, the author believes that a more proper name for this condition is myotonia hereditaria rather than myotonia congenita.

Etiology: Numerous investigators have proposed theories as to the explanation of this condition: (1) cerebrospinal genesis (Thomsen, Engel, Curschmann), (2) myopathic theory (Strümpell, Nissen), (3) chemical toxic theory, (4) internal secretion theory. The two latter theories are mentioned but have comparatively few followers.

Prognosis and Therapy: Briefly stated, the prognosis quoad vitam is good, quoad sanationem, absolutely unfavorable. Glandular therapy in all forms has been tried out, but with uniform failure. Exercise, which has been recommended from the time of Thomsen, should be avoided. Unfortunately, no complete pathologic studies have been made by the author, and he hopes that in time further progress may be made following such investigation.

Summary: In summary, the author makes the following sixteen points:

1. Leyden deserves priority in describing Thomsen's disease.
2. Bell has described no case in his works.
3. Paramyotonia congenita and myotonia congenita intermittens belong to a disease group for which the term paramyotonia hereditaria should be used.
4. Myotonia aquisita is not recognized as a disease entity, but probably represents a latent form of Thomsen's disease.
5. Myotonia atrophica should be considered as a disease entity.
6. Intention rigidity may be observed in the early years of childhood in Thomsen's disease.
7. The occasional beginning of intention rigidity only after several movements must not be taken as a paradoxic form of Thomsen's disease.
8. Psychic disturbances may accompany the picture of Thomsen's disease, but do not belong to the sydrome.
9. Myotonia congenita follows the mendelian law of heredity.
10. From the ninth point, it follows that eugenic measures should be instituted in cases of Thomsen's disease.
11. The hereditary factor is always assumed to be present in this disease.
12. There are too few cerebrospinal findings to warrant such a cerebrospinal theory.
13. Myotonia congenita must be considered as a myopathy, and the causative factor may rest in some chemical change of the sarcoplasm or Keimplasm.
14. Prognosis quoad vitam good, quoad restitutionam unfavorable.
15. Therapy thus far has been unsuccessful.
16. It was suggested that the term myotonia hereditaria be used in place of the term myotonia congenita.

Nissen adds an exhaustive bibliography of over 170 references.

MOERSCH, Rochester, Minn.

THE OCCURRENCE IN OSTEITIS DEFORMANS OF LESIONS OF THE CENTRAL NERVOUS SYSTEM, WITH A REPORT OF FOUR CASES. W. G. WYLLIE, Brain **46**:336 (Oct.) 1923.

Despite the changes in the bone surrounding the brain and spinal cord in osteitis deformans (Paget's disease, 1876) the nervous system is rarely affected. Hurwitz found the sella turcica altered in one case of six which he studied. Leri and Jefferson both mention cases in which the sella turcica had become shallow. The central nervous system changes are not mentioned.

Four cases are reported, the first two with compression of the spinal cord. Case 1 was that of a man, aged 55, who a year previously had lancinating pains in the buttocks on walking, cold feet and difficulty in locomotion. At

the end of a year he could just shuffle along with the aid of two sticks. Six months previously, he had noticed that hot water felt cold to his feet. He had frequency of urination and rectal incontinence after purging. Physical examination revealed bony changes in the skull typical of Paget's disease. On the trunk, below the third costal cartilages, there was impairment to a moderate degree of all forms of sensation. Both legs were paretic, with no muscular wasting. The abdominal reflexes were absent. There was a bilateral Babinski sign, and the deep reflexes of the lower limbs were exaggerated. Lumbar puncture was unsuccessful. Roentgenograms showed typical changes in the skull. Because of the gradual progression of symptoms, the patient was operated on, and a laminectomy made from the second to the fourth dorsal vertebral arches. The bone was thickened, softened and cut easily. The perithecal space was absent, and the inner surfaces of the laminae were closely opposed to the dura mater. The patient made a good recovery and left the hospital after two months, walking with much more ease than on admission. Microscopic examination of the bone showed changes typical of osteitis deformans.

Case 2 was that of a man, aged 63, who since the age of 49 had suffered with sciatica of both legs. The pain was accompanied by spasms and cramps in both legs, and he found difficulty in controlling his urine. At 61, he noticed that his feet became numb and blue, his legs were unsteady, and he fell down on several occasions. After a year his legs felt heavy, he had a tight feeling around the upper part of his abdomen, and paralysis finally became complete in his lower extremities. He had "precipitant micturition," and rectal control was defective. Physical examination revealed complete sensory loss to all stimuli over the trunk and lower extremities. Vibratory sense was absent below the level of the seventh dorsal spine. The ankle reflexes were exaggerated, the knee reflexes were difficult to obtain, and there was a bilateral Babinski sign. The abdominal reflexes were absent. Lumbar puncture showed a scanty yellow fluid, which coagulated on standing. Roentgenograms suggested a tumor at the level of the fourth and fifth dorsal spines. Necropsy showed a narrowing of the vertebral canal due to thickening of the vertebrae, with a resulting compression of the cord. The main compression in this case, however, probably came from bony excrescences projecting from the inner surfaces of the bones. There were no changes suggestive of osteitis deformans in any of the other bones of the body.

The author mentions the possibility of compression of the spinal cord in osteitis deformans in two ways: (1) by a narrowing of the vertebral canal with resulting compression of the cord; (2) by bony excrescences which project on the cord. The author studied the vertebral column of a cadaver in which there was osteitis deformans of the skull, facial bones and vertebrae. He found a distinct narrowing of the canal, especially noticeable in the lumbar region. There was, moreover, an irregularity and lipping of the edges of the vertebrae.

Marie and Leri report a case of osteitis deformans with a syringomyelic cavity from the first cervical to the second dorsal segments of the cord. Touretti and Marinesco report a case in which the symptoms may have been due to compression of the cord by bony changes.

Two other cases are reported in which atrophy of the optic nerve occurred in skulls affected by osteitis deformans.

Case 3 was that of a woman, aged 61, who at 53 was troubled with diplopia. This disappeared in six months. At 60, her right vision began to fail, and she

was found to have atrophy of both optic nerves. Examination by roentgen ray showed a thickening of the skull bones. The right disk was pale, the visual acuity was 6/12 in each eye, and a large irregular scotoma was found in the right eye, with some constriction of the field. The left visual field was normal. Case 4 was similar.

"It appears permissible by a process of exclusion, to assume that in these two cases of osteitis deformans the atrophy of the optic nerves resulted from their compression within the optic foramina. The state of the pupil reactions, and the absence of any clinical signs of tabes dorsalis, were evidence against the condition's being of luetic origin. The possibility of sinus infection could be set aside by the examination of the skiagrams of the skulls.

"Clinical histories failed to suggest the existence of any other possible cause of optic atrophy, whether toxic, infective or familial."

An examination of two skulls from cases of osteitis deformans showed some narrowing of the optic foramina. The capacity of the eye sockets was also considerably reduced. Paget mentions blindness in four cases of his twenty-three. Coppey reported four cases with degenerative changes in the retinae. Vergue reports three cases with well marked chorioretinitis. Crystalline deposits in the lenses have been observed by many.

ALPERS, Philadelphia.

THE REMISSION OF MENTAL DISORDERS JUST BEFORE INTERCURRENT SOMATIC DISEASES. PAUL COURBON, Rev. neurol. **30**:237 (March) 1923.

The writer recalls the fact that it has long been recognized that in certain patients suffering from mental disorders there appears a remission, with perhaps a complete restoration of reason, just before death or in the course of an intercurrent febrile disease, or even in certain afebrile diseases such as ulcer of the stomach and cancer. This has been recognized from the time of Regis to Kraepelin. A disappearance of delusions in paranoid cases may occur just before death, or mental symptoms in a dementia praecox case may cease in the course of a pulmonary tuberculous episode. Such remissions over a shorter or a longer period of time may occur in both the acute and in the chronic psychoses. It is such remissions in general paralysis under the influence of acute infections that have inspired the serotherapeutic measures sanctioned by Robertson, Levis Bruce and Wagner von Jauregg.

The apparent antagonism between physical and psychic integrity may be due, according to M. Leroy, to the "humoral" modifications caused by the intercurrent affection of one or many organs. The mechanism of this action is still a mystery; nevertheless, one can formulate certain suggestions, e. g., among certain psychopathic persons, healthy psychic neuron patterns, and diseased neuron patterns function alternatively, the activity of the first having for its existence the presence of an alteration of the corporal health.

We know that a profound functional disturbance may occur, in spite of the integrity of the majority of anatomic elements in a given system, if one of these elements be injured. As regards the nervous system, we are acquainted with von Monakow's theory of diaschisis, a diaschisis which is only ephemeral, according to the Swiss neurologist. The war taught how much more intense a functional disturbance may be than we would expect from the comparatively limited extent of the actual lesion. This is illustrated in Babinski and Froment's studies in hysteria and reflex disturbances. Pic (1915) says: "For a long time I have come to believe, based on a series of autopsies, that a very

limited lesion may have an enormous importance, not only in its bearing on the nervous system, but also in its effects on the kidneys, the heart, and most of the viscera."

There is raised, then, a rather daring question: Why may not the viscera and motor portion of the brain also have sentient and thought potentialities? Of course it is recognized that there is no "center" for intelligence, emotion or will; on the other hand, the psychic processes are apparently a result of synergy between various cerebral areas. Why, therefore, should not an injury to certain neurons whose function it is to effect this synergy also inhibit the action of areas spared? It apparently does so completely in certain persons. This first hypothesis of the inhibiting power of healthy psychic neurons by diseased psychic neurons whose activity results in a vicious function appears contradictory to the established clinical and anatomopathologic facts of general pathology. It therefore remains to be explained how there is a complete reversal in the sick body, that is, an inhibition of the disease processes and an activity instead of the healthy elements.

The first attempted answer is that there may be a parallel here to the conditions existing when we attempt curative process by serums, such as obtain in diptheria and certain fevers.

The diseased or disordered psychic neurons, by the very fact of their morbidity, are much more vulnerable than healthy psychic neurons; wherefore their *paralysis* and in consequence the lifting of the inhibitions which they exercise on other neurons. The latter lie dormantly healthy, and with the integrity of their synapses they may resume their healthy activity. They reestablish between various cerebral areas pathways of communication whose visibility may bring about the disappearance of perverse emotional states, errors of judgment, faults of memory, etc. If before the cure the organic malady has had a chance to destroy the psychic disturbance, the mental trouble will no longer reappear, and the cure will be final. Such a conception whether confirmed by necropsy or not must conform to the reality of the facts. Its principal merit is that it gives us an explanation of a clinical cure not accompanied by an anatomic cure. A disordered function ought to have for its essential cause the vicious action of diseased portions of the organ and not the inertia of dead elements of that organ. If it be true, as certain writers have maintained, that dementia can be cured, it is without doubt because the vicious functioning of irremediably changed cells has been substituted for by that of healthy cells, not newly formed ones, not by the formation of new parenchyma, but rather through the complete inhibition of disordered cellular action.

The fact that all psychopathic persons are not helped by bodily disease may be explained by the qualitative difference in the cerebral substance. What is yet needed, and what may take a long period of time to discover, is the determination of the exact dose necessary to bring about the definitive death of diseased neurons, at the same time sparing the healthy neurons, and thus furnishing the cure.

JONES, Detroit.

A HITHERTO UNDESCRIBED PUPILLARY REFLEX (PHARYNX REFLEX OF THE PUPIL). R. FINKELNBURG, Ztschr. f. d. ges. Neurol. u. Psychiat. **91**:183 (June) 1924.

The author describes a case with a pupillary phenomenon which has not hitherto been described. The case was that of an 18 year old boy, who at the age of 13 or 14 developed dilatation of his right pupil without other

symptoms. At the age of 15, a physician was consulted for the condition, and at that time there were found a dilated right pupil, normal fundus, normal vision and accommodation limited on the right side. At 18, the author saw the patient, who at this time complained of a feeling of cold and of a reddish blue discoloration of the left hand and forearm, gradually progressive for two years and worse in cold weather. He complained also of tingling in the little and ring fingers of his left hand in cold water, and of stiffness of his left hand in playing the piano. Examination revealed: the right pupil twice as wide as the left, the reaction to light lost on the right but prompt and full on the left, accommodation slight on the right but normal on the left. The consensual reaction was absent in the right pupil, but present in the left. Tactile stimulation of the conjunctiva and cornea produced immediate contraction of the right pupil, lasting five or six seconds, followed by a slow dilatation in the next ten or fifteen seconds. Tactile stimulation of the left cornea produced definite but slight contraction. Painful stimuli to the right and left conjunctivae caused contraction of both pupils. Tactile stimulation of the posterior pharynx produced a maximal contraction of the right pupil, while the left stimultaneously became strongly dilated. The same phenomenon occurred on drinking warm water. Swallowing cold fluids or nothing or well-masticated bread produced no change in the pupils. The other neurologic signs consisted of arm reflexes more active on the right than on the left, normal knee reflexes, no ankle clonus or Babinski sign, and a disturbance of pain and temperature sensation over the left shoulder, outer side of the left arm, the entire forearm and most particularly over the left hand. In 1923, at the age of 20, a nystagmus developed.

The author considers two possibilities in the explanation of the pupillary phenomena: (1) the contraction depends on a physiologically simultaneous movement of the pupil analogous to the pupillary contraction in closing the lids, and is therefore only a concomitant symptom of the pharynx reflex; (2) or the condition is a true trigeminal reflex contraction (Trigeminusverenger-ungreflex), which under normal conditions does not appear because of the predominance of the sensory dilatation reflex, but which does appear under pathologic conditions.

The assumption of a simultaneous movement of the pupil at the time of the pharyngeal reflex presupposes that the stimulus to the pharyngeal muscles is propagated, directly or indirectly, to the nucleus of the oculomotor nerve. The same assumption is made in the contraction seen on closing the eyelids. On this basis, it might be assumed that in both of the foregoing cases we have a reflex process, with the difference that the sphincter nucleus is not stimulated directly, but is innervated through a motor neuron between the nuclei of the vagus-glossopharyngeus and the oculomotor. The posterior longitudinal bundle would appear to be the connecting system or link. There are certain objections to this theory, however. In the first place, it is striking that the reflex does not occur in the normal person. Again, if the reflex is dependent on a contraction of the pharyngeal musculature, we should get a contraction of the left pupil also; instead we get a dilatation. The occurrence of the phenomenon only with the swallowing of warm fluids and not with cold or with a voluntary contraction of the pharyngeal musculature would throw doubt on the foregoing theory.

The author suggests the possibility that we are dealing here with a true sensory reflex contraction of the pupil. As support for this, he brings the

fact that maximal miosis occurred not only through stimulation of the pharyngeal mucosa, but also through the tactile pharyngeal mucosa, and also through tactile stimulation of the conjunctiva and cornea. The theory that the author finally reaches in explaining the foregoing pupillary phenomena is that physiologically the dilator and sphincter muscles of the eye are brought into action with every trigeminal stimulus. Normally, the dilator reflex is stronger than the contractor. A "reflexumkehr," or an abnormal prodominance of the contractor reflex, can be produced, however, if the normally dominating influence of the dilator reflex is side-tracked. This is made possible by disease processes, which either harm the centers and paths of the dilator reflex, or set the ganglion centers of the contractor reflex into a condition of increased excitability. This may be produced by direct stimulation of the ganglion cells through neighboring foci of disease processes indirectly. What the diagnostic value of the sign may be, is hard to say. Further observations are necessary in order to draw any conclusions on this side of the question.

ALPERS, Philadelphia

A SUPPLEMENT TO THE AUTHOR'S AUTO-OBSERVATION ON THE PSYCHIC AND NERVOUS ACTIVITY FOLLOWING CEREBRAL THROMBOSIS (OR APOPLEXY) DURING MAY AND OCTOBER, 1912. A. FOREL, J. f. Psychol. u. Neurol. **30**:162, 1924.

This paper should be read in conjunction with the previous one published by the author in this journal (**21**:417, 1915). In this supplement, Forel corrects some of his observations published in 1915, especially as to the various degrees of consciousness. There exist quantitative and qualitative degrees of consciousness. The vividness of the momentary central point of the attention affects the clearness of consciousness or the introspection, which, as Semon has shown, is not of the same significance as the intensity of consciousness. Vividness of consciousness gives rise, above all, to clearness of detail. But a confused engram complex may also be intensive. Associated with intensiveness, vididness mostly affects the intensity of introspection or consciousness by means of the faculty of attention. An association of this nature Forel designates as the *first* maximal degree of the quantity and quality of consciousness. Following the same line of reasoning he would designate marked vividness and slight intensity as the second degree of consciousness (while listening carefully and attentively to pianissimo). Marked intensiveness and slight vividness are characteristic of the third stage (intense but dull, muffled noise). In this stage, one may also listen attentively, but the tones are confused and are not sharply distinguished from one another. This may be due to external factors in the production of the tones, and also to internal factors in our physical and psychic audition or in some of the other senses. As a fourth and more defined stage, he would designate states of consciousness that are neither vivid nor intense. They are, so to say, within the periphery of attention, and, though somewhat flighty, they can still be perceived. Examples of this are furnished by parts of the visual field situated beyond the macula lutea. The same may be said of the sensations of hearing and taste, and to a lesser degree of smell, temperature and pain. In this stage, the perceptions are still well associated but are only temporarily and feebly brought to life again, because the faculty of attention is occupied somewhere else. This fourth stage is therefore automatic in nature, occupying old traveled pathways, and repeated in a mechanical and parrot-like fashion. It sinks deeper and deeper into a well ordered form of secondary subconsciousness—secondary automatism (recalling semiconsciously some old

melody while thinking of something else). The fifth stage is that of somnolence or slumber. This stage marks the beginning of a principal qualitative difference—a genuine subconsciousness. The faculty of attention generally declines, and this gives rise to a dissociation of thinking and of the affective life, with its corresponding parekphorias; half dreams and dreams, and with these a relative rest of cerebral activity, are also characteristic of this stage of consciousness. Depending on the degree of slumber, hallucinations and illusions also appear in this stage. The fifth and sixth stages are of great significance in hypnosis and psychanalysis. The sixth stage is encountered in severe cases in which long chains of reminiscences or engram complexes appear to be entirely forgotten. In these cases, as well as in many others subjected to psychanalysis, it is found that the ekphoria (ability to recall to memory) of important engram complexes or even of entire parts of the person's life, becomes unusually difficult, and can only be accomplished by hypnosis or by a method of psychanalysis that requires unusual patience and a special technic. The seventh stage is one in which forgetting is absolute and definite, no matter whether it concerns dreams, hynosis, or life experiences while awake. We all forget a considerable part of the processes of our existence that have been engrammed in our brain. Nevertheless, this sixth stage, as well as Semon's researches, clearly shows that apparently forgotten engrams (memory pictures) can remain in the brain —and this, even though the neurons have not been injured. In the latter instance, we naturally get a definite amnesia. The so-called seventh stage can still be said to belong to the cerebral cortex. Other stages are in existence, but they concern purely theoretic and hypothetic considerations based on observations and experimentation. Comparative anatomy and psychology, however, compel us to accept them. Forel would consider the subcortical engrams of the so-called basal ganglia, and of the cerebellum and cord, as the eighth stage. Subjectively, however, nothing definite is known about these phenomena. As a ninth stage, one can assume hypothetically the existence of a consciousness in an individual ganglion or ganglion cell. And, finally, as a tenth stage, one can think of the existence of animals that have no nervous systems (amebas and protozoa). The cells of plants could be included in this stage.

KESCHNER, New York.

A STUDY OF THE PATHOLOGIC PHYSIOLOGY OF THE CEREBRAL VENTRICLES IN MAN. CESTAN, RISER and LABORDE, Rev. neurol. **30**:353 (April) 1923.

The authors state that they have performed 150 ventricular punctures according to an exact technic arrived at by study on the cadaver. It is their experience that ventricular puncture is practically free from all danger.

In the case of the normal person seated or lying directly on his back, intraventricular pressure is practically nil. It is necessary to aspirate the fluid. The plunger of the syringe returns spontaneously into the barrel even when one aspirates as much as 5 or 6 c.c. of fluid.

A study of the chemical constituents of the ventricular fluid gives the following interesting results as compared with that obtained by the classic puncture of the spinal canal. The total amount of albumin and the number of cells is very low, but in the majority of cases the sugar is high, e. g., 60:45 is the ratio reported.

The rest of the paper is divided under four heads, namely: (1) experimental modifications of intraventricular pressure, (2) permeability of the lateral ventricle after intravenous injection, (3) relations between the spinal canal and the cerebral subarachnoid spaces—ventricular emptying, and (4) relations

between the ventricular space and the spinal subarachnoid spaces. Considering each briefly in order, the following conclusions are arrived at:

1. In the horizontal or the sitting position, the pressure of the intraventricular fluid is 0. Its pressure is sharply increased up to 3 or even 15 c.c. (water pressure) by lowering the head in the direction of an inclined plane, this increase being brought about by a flow from the subarachnoid spinal spaces or by an increase of the volume of the encephalon in the strictly inelastic cranial vault or skull under the influence of the engorgement of the encephalic veins. The same result is obtained even with the patient in the sitting position by exerting pressure on the jugular veins or by the inhalation of amyl nitrite. Theobromin, pilocarpin, and caffein apparently have no effect on intraventricular pressure; epinephrin increases it for from ten to fifteen minutes; but the latter seems to have its effect on arterial pressure in the region of the choroid plexus.

2. In the normal state, iodid of potassium and methylene blue injected intravenously does not traverse the barrier set up by the choroid plexus, but if the secretion of the choroid be modified by the intravenous injection of extract of choroid tissue, there will quickly be found small quantities of potassium iodid and methylene blue in the ventricular fluid. The same results obtain when from 20 to 30 c.c. of spinal fluid are withdrawn. This is interesting on its bearing from a therapeutic point of view relative to the forcing of medication past the choroid plexus into the ventricular spaces.

3. Neutral red in an isotonic solution (1:20), may be found in appreciable quantities sufficient to show color effects at the level of the fissure of Rolando (subarachnoid spaces) within twelve hours after injection.

4. This topic is discussed under two subdivisions (relations between the ventricular space, and the subarachnoid spinal spaces).

(a) Descending Ventriculospinal Relations.—In a normal person, strictly immobile and placed in lateral horizontal decubitus, breathing regularly, not coughing, an injection of 1 c.c. of neutral red into the lateral ventricle (after withdrawing an equal quantity of ventricular fluid) colors the spinal fluid in the lumbosacral region only faintly after an interval of two or three hours. If, however, the patient sits up, walks, or coughs, the spinal fluid rapidly shows red. The same result is obtained if a certain quantity of spinal fluid is withdrawn, or if extract of choroid tissue is injected. The application for therapeutic purposes is self-evident.

(b) Ascending Spinoventricular Relations.—In the normal state, substances injected in small quantities (0.5 to 1 c.c.) in the lumbosacral spinal cavity do not rise to the cerebral subarachnoid spaces. Putting the patient on an incline, head lowered, however, produces different effects, as has been demonstrated by cocainization of the spinal fluid. It is for this same reason that more lymphocytes are found in the spinal fluid of tabetic persons than in the ventricular fluid. Consequently, if one wishes to get therapeutic substances into the cerebral subarachnoid spaces, it becomes necessary to go through the following procedure: (1) As a preliminary measure, draw off about 30 c.c. of spinal fluid, mix the desired substances in this fluid, reinject the fluid with the medicine mixed therein; (2) lower the patient's head, the body on an incline.

The writers conclude with the statement that in certain cases ventricular puncture is superior to lumbar puncture from the point of view of the therapy which has in mind treatment of the meningeal spaces or the central nuclei.

JONES, Detroit.

THE PHYSIOLOGY OF THE HAND FOREARM REFLEX. KARL FLICK, J. Psychol. u. Neurol. **29**:93-111, 1922.

Leri, in 1913, described the sign of the forearm as follows: The patient relaxes the arm; the forearm is supported by the examiner's left hand and with the right hand the fingers are bent toward the hand, the hand against the forearm, causing the hand to roll into itself. With this action the forearm flexes itself slowly. The flexion continues as long as the fingers and hand are held in this position. At its maximum, the hand will be a few centimeters from the shoulder.

This is supposed to be a physiologic reflex, and it disappears only in certain pathologic cases. In normal persons, it is always present although not to the same degree. It is rarely exaggerated, but may appear diminished. The side on which it is absent is the affected side.

Leri considers the reflex arc to be the radial and musculocutaneous nerves, the pyramidal tracts and the motor cortex. A lesion in a portion of this arc will cause the reflex to disappear. Both Leri and Gurewicz have come to the conclusion that this reflex is either diminished or absent in the following conditions: (1) tabes of the upper cervical region; (2) hemiplegias; (3) cerebral diplegia; (4) Friedreich's ataxia; (5) Amyotrophic lateral sclerosis; (6) occasionally in cerebral tumors; (7) usually in syringomyelia, multiple sclerosis, cord tumors of the cervical region; (8) in most cases of cerebral syphilis or spinal syphilis; (9) usually in Huntington's chorea and epilepsy; (10) brachial plexus neuritis, muscular atrophy, poliomyelitis.

The reflex is present in: (1) vasomotor neuroses, (2) progressive paralysis, (3) paraplegia, (4) cerebral diseases and (5) some cerebral traumas.

During the past few years, other observers have worked with this reflex. Mayer found it absent in ten of eighty-two normal persons. Goldstein obtained a positive in 98 per cent. of 1,000 cases. He also noticed its absence prior to the second year. Both Mayer and Goldstein consider the reflex a proprioceptive reflex with a cortical reflex arc.

Flick, in analyzing this reflex, states that it does not compare with any other neurologic reflex, and he wonders whether the natural contraction of the hand would not cause a bending of the elbow or even that flexion be due to the examiner's pressure. He tried out the reflex on a large group of normal persons and usually found it present. In the cases in which it was absent it was found that the patients were loose jointed or had nimble and pliable fingers. He believed that the reflex was probably the result of mechanical conditions, and that when pressure was made on the extensors of the fingers, the resulting effect would be a flexion in the elbow, as the extensors were too short to stand this extra pressure. Leri, on the other hand, is of the opinion that the reflex is a defense mechanism. Flick, while agreeing with this, does not believe that it is a true reflex. Working with patients and his colleagues, he found that they all complained of pain, and he received the impression that the movements took place as the result of this pain. In cases in which the sign is absent in normal persons, the author believes that it is due to a voluntary inhibition on the part of the patient, just as a limb may be held quiet in spite of some slight painful operation. This is not the only phenomena of this type, but as noted by Flick, practically any painful procedure, such as the rolling inward of the pronated hand, will cause a lateral elevation of the upper arm and shoulder joint. These reactions, while reflex, are not to be compared with the accustomed physiologic reflexes.

The author by experimental work has come to the conclusion that this reaction is the result of muscular response and not part of a reflex mechanism. He believes that there are three reasons for the absence of the hand-forearm sign in cases of hemiplegia: 1. There is impairment of motion on the affected side. 2. There is impairment of sensation on the affected side. 3. There is lack of proper tonus and diminished pain reaction. For any one of these three reasons the reflex may be absent in such conditions as syringomyelia, multiple sclerosis, tabes and the like.

In conclusion, the author states that in certain cases of pyramidal affection the hand-forearm sign is not obtained, but that its mechanism is not a purely physiologic one, and that it must not be considered as possessing any definite clinical value.

MOERSCH, Rochester, Minn.

ANXIETY CONDITIONS AND THE VEGETATIVE NERVOUS SYSTEM. ERICH DAVID, Ztschr. f. d. ges. Neurol. u. Psychiat. **91**:209 (June) 1924.

Freud pointed out that anxiety is connected with the sexual instinct. Sexual gratification, for example, can offset anxiety under certain conditions, and anxiety can be converted into other syndromes, such as asthmatic attacks, diarrhea, vertigo, etc. In many cases, moreover, in which an anxiety condition appears in the psychosis, there is a tendency to practice onanism.

The present-day psychiatry directs its attention more to the vegetative disturbances that occur in the course of mental upsets, and it is from the point of view of the vegetative nervous system that the author attacks the problem of anxiety states. The similarity of the condition of exophthalmic goiter to the anxiety neurosis of Freud has long been apparent. In both conditions there is predominant an anxious, irritable, excitable disposition, and both show palpitation, vertigo, outbreaks of perspiration, diarrhea, congestion and a tendency to polyuria and glycosuria. It would seem as though exophthalmic goiter is only an aggravated form of anxiety neurosis, and it must be conceded that the conception of "morbus Basedowie" as a constitutional neurosis has much for it both theoretically and clinically. Möbius pointed out also that a number of symptoms of exophthalmic goiter, namely, exophthalmos, palpitation and tachycardia, tremor and perspiration are encountered also in an active anxiety condition. We know, too, that variations in temper influence the secretion of the internal glands; for example, thyroid secretion is influenced by increased excitability and vice versa.

The similarity of exophthalmic goiter and anxiety conditions is quite apparent. In exophthalmic goiter we have tachycardia, enlargement of the thyroid, exophthalmos, perspiration, disturbances of digestion, ravenous appetite, diarrhea, vomiting, tremor and even a peculiar paraparalysis of the legs. Freud gives the following as characteristic of anxiety conditions: (1) palpitation of the heart with slight arrhythmia, long-sustained tachycardia which may cause decompensation, and pseudo-angina pectoris; (2) disturbances of breathing, several forms of nervous dyspnea, asthmatic attacks, etc.; (3) perspiration; (4) attacks of shaking and trembling; (5) diarrhea; (6) vertigo; (7) attacks of so-called congestion; (8) attacks of paresthesia.

If we consider the physical disturbances that occur in exophthalmic goiter and in anxiety neurosis, we find that they are due to an increased sympathicotonus, but more especially to an increased parasympathicotonus. Since we have in both conditions similar psychic and physical disturbances, we must

expect the cause of the trouble to be the same in both conditions. As the cause of exophthalmic goiter we look on disease of the thyroid gland, while in anxiety neurosis we have a connection with sexuality.

Certain evidence has shown that the thyroid and sexual glands stand in a certain definite relationship to one another, and it is therefore not to be wondered at that we have similar pictures in exophthalmic goiter and in anxiety neurosis. Fisher showed that the thyroid gland swells during puberty and in menstruation, and that all the symptoms of exophthalmic goiter are apparent before and after menstruation. Kron concluded that exophthalmic goiter causes secondary changes in the sexual life of the woman. Gruber found a small thyroid gland in a eunuch. Tandler and Gross found a myxedematous type in the aged. Fliesz asserted that he was able to check the vomiting of pregnancy by the administration of thyroid tablets. There is a direct dependence of the endocrine system, the vegetative system and the nervous system on one another. The author asserts that the endocrine and vegetative systems, acting independently or in unison, affect the vegetative center in the midbrain, and in this way influence the mind by a direct effect on the metabolism of the individual. It is by producing an increase or a decrease in the metabolism that the vegetative nervous system has its effect on mental reactions, and particularly in conditions of anxiety.

ALPERS, Philadelphia

THE TREATMENT OF SPASTIC PARALYSIS. CHARLES E. DOWMAN and MICHAEL HOKE, Arch. Surg. **9**:145 (July) 1924.

One hundred and thirty-two cases of infantile cerebral paralysis are considered in this study. The etiology producing this condition is often difficult to determine. Prenatal, natal and postnatal factors are at work. In thirty-two cases it was impossible to assign any definite cause for the development of the condition. Thirty-six cases were classified as being due to prenatal faults, syphilis, hydrocephalus and encephalo-meningitis being the more important. In twenty-nine cases, the spastic paralysis seemed to be due to birth injuries. Thirty-seven cases were produced by postnatal conditions; in sixteen, convulsions of undetermined etiology were followed by hemiplegia; in eighteen, a febrile disturbance occurred with subsequent paralysis; and in three, a definite meningitis preceded the onset of the spastic paralysis.

Clinically, this series of cases is grouped into three divisions: a group in which the paralysis is of the pyramidal tract lesion type, eighty-four cases; a second group, in which the cause was apparently an extrapyramidal lesion, thirty-one cases; a third group, in which both pyramidal and extrapyramidal tracts are involved, eleven cases. Only those cases belonging to the pyramidal tract group are suitable for treatment. All cases of extrapyramidal or of the mixed type were discarded, for these cases are not true spastics, and there are no particular groups of muscles which could be relaxed in order to obtain proper muscular balance. In the second place, should one correct through operative measures the various deformities that may be present, the child would still be unable to perform coordinated muscular movement, as the coordinating mechanism is interfered with, and therefore no practical results could be expected. The cases selected for treatment must be true spastics due to lesions of the pyramidal motor system. As physical training is one of the most important features of the treatment, there must of necessity be suffi-

cient mentality of the individual patient to permit cooperation as well as endeavor on the part of the patient in making the proper effort to walk or to utilize an involved upper extremity. If this mentality is not present, it is futile to attempt to give muscles the proper balance or to correct existing deformities, as the child will be unable to accept the necessary physical training. We have selected arbitrarily a mental age of 4 years as a minimum degree of mental development necessary if any favorable results are to be expected.

The method of treatment which has been used in a limited number of cases consists of three important features: (1) an attempt to restore proper muscular balance by weakening through neurectomy those groups of muscles which outweigh in muscular strength the opposing groups; (2) the correction, through orthopedic procedures, of certain existing deformities which have resulted from long standing malposition of the joints, and the stabilization of the feet, which, as the result of the interference with proper cerebral control cannot be held by the patient in the necessary position suitable for proper weight-bearing organs; (3) proper training by one thoroughly versed in this line of work, in order to teach the patient to utilize the extremities after the muscular balance has been restored and the existing deformities have been corrected.

Muscular relaxation was obtained by partial neurectomy of the nerves supplying the muscles at fault as suggested by Stoffel. The orthopedic procedures were many and varied as conditions required and played an important part in the treatment. It would be useless to relax a child's muscles by neurectomy unless his extremities were in condition to bear his weight. In all, eighteen patients were treated in this manner, and the results obtained seem to justify the opinion that this combination of methods of treatment is essential if the spastic child is to be given the maximum amount of useful function.

Detailed case histories and descriptions of the different procedures employed are included in the article.

GRANT, Philadelphia.

THE TREATMENT OF GENERAL PARALYSIS WITH MALARIA. ILSE GRAF, Ztschr. f. d. ges. Neurol. u. Psychiat. **91**:131 (June) 1924.

The good results reported from other clinics in the treatment of general paralysis with malaria, and the good and long remissions which are obtained, made an investigation of the malarial treatment of paralysis worth while to the author. The technic followed consisted in the injection of 0.75 c.c. of malarial blood obtained at the height of the fever, and the administration of quinin hydrochlorid after the tenth febrile attack (1 gm. for three days, and 9.5 gm. for from two to three weeks). The remissions were divided as follows: (1) complete remissions in which the patient has no more noticeable defects, and also a return of working capacity; (2) incomplete remissions in which are included cases in which there is a return of earning capacity but still visible defects, and also cases in which the patients, although not entirely well, are better than before treatment.

Patients who were first treated in 1921 have been followed for over two years. There are seven such cases. Of these seven, remissions occurred in five. Of these five, one was complete and four incomplete. Of the remaining two cases, one patient died and one was unimproved. Some doubt was felt as to whether this last case was a true paralysis or whether it was a case of chronic alcoholism. The history of an infection, the clinical picture and the positive blood and spinal fluid serology, however, all pointed to paralysis.

In 1923, twelve other patients were given malarial treatment. Of these twelve patients, two showed definite remissions, and two became more active and interested in their surroundings, after the conclusion of the treatment. Both showed marked defects, however, and the author suggests the possibility of beginning remissions in these cases. One patient developed an icterus before the onset of the fever. Two other patients died, and five cases remained unchanged.

The author draws the following conclusions from his study of his nineteen cases: 1. Slightly advanced cases with slight intellectual defects are the most favorable for treatment with malaria. 2. Acute cases of paralysis with expansive grandiose ideas are particularly favorably influenced by malarial treatment. 3. No deaths occurred as a result of the malaria. In both cases in which death occurred, there was no fever, and no parasites could be found in the blood. 4. In two cases, a combination of malaria and arsphenamin was tried, in one of them mercury also being used. Both these patients have had good remissions, the one receiving the mercury in addition to arsphenamin and malaria being particularly well. 5. In two cases, there was a remission of two years; in one case, there was a remission of one and one-half years. 6. The spinal fluid of five patients with pleocytosis showed a return of the cell count to normal. The blood Wassermann test became negative in two cases. The spinal fluid Wassermann test became negative in one case.

The author calls attention to the small number of cases presented—nineteen in all, and also emphasizes the fact that remissions occur "normally" in general paralysis. He thinks, however, that further treatment with malaria is justified.

ALPERS, Philadelphia.

KLÄSI'S PERMANENT NARCOSIS IN TWO CASES OF MANIC-DEPRESSIVE PSYCHOSIS (PRELIMINARY NOTE). J. M. SACRISTÁN and J. G. PINTO, Arch. de neurobiol. **4**:139-150, 1924.

This is a detailed report of two cases of severe manic-depressive psychosis in women, successfully treated with injections of somnifen. Kläsi's directions were closely followed.

CASE 1.—A married woman, aged 40, with seven children and four abortions, entered the asylum (where she had been twelve years before) after a period of excitation and intense jealousy caused by conjugal infidelity. Somatic and neurologic examinations were negative; the urine, heart and circulation were normal. In the asylum, she suffered a severe attack of typical mania; she spent two nights singing until she became hoarse. A permanent bath did not produce any improvement. During the following four days, she sang and laughed, showing intense flight of ideas, coprolalia, aggressiveness and manifest distraction. She was injected with somniferin for four consecutive days (two injections daily on the first three days, one injection on the fourth day; treatment began with an injection of scopolamin and morphin). Treatment was interrupted during menstruation. A slight improvement was followed by a new attack of depressive mania. Injections of somniferin were again given for five days. At the end of this period, marked improvement was manifested in completely normal behavior, increase in weight and normal sleep and appetite. She was discharged a month later.

CASE 2.—A married school teacher, aged 47, with six children and two abortions, entered the asylum during a period of intense excitation with deep psychomotor agitation. She laughed and sang, talking continuously. Neurologic

and somatic examinations were negative. She suffered from mucous membranose colitis and had undergone an operation of uterine curettage. Between attacks she behaved normally and manifested intelligence and clear judgment. Since the attacks were frequent, treatment with somniferin was begun; she received injections of this substance for ten consecutive days, followed by marked improvement. A new attack, not so intense as the preceding, less than a month after first treatment, required more injections for seven days, with successful results.

The authors discuss the value of Kläsi's method in the treatment of manic-depressive psychosis, and point out that in contrast with schizophrenia, the use of subsequent psychotherapeutic methods is unnecessary. They emphasize the fact that the treatment is by no means curative, but that it merely supresses the manic attacks. From their experience, they conclude: 1. Kläsi's method may yield excellent results in the practice of psychiatry, especially in attacks of manic-depressive psychosis, shortening their duration to a minimum, which the ordinary methods followed in psychiatric clinics seldom attained. 2. It is possible that Cloetta's hypothesis is fulfilled in cases of manic-depressive psychosis, and that perhaps these cases are the best adapted for this type of treatment. (3) Permanent narcosis applied, following the technic indicated by Kläsi, does not involve any danger to the patient.

NONIDEZ, New York.

THE BLOOD SUGAR TOLERANCE IN PSYCHOSES. SHOZO TSUCHIYA, Ztschr. f. d. ges. Neurol. u. Psychiat. **91**:23 (June) 1924.

Many investigations have been carried out since the introduction of microscopic methods for the determination of the blood sugar. Myers, in a case of thrombosis, and Krehbial, in cases of carcinoma, noticed a marked change in the sugar content of the blood. Mita first studied the sugar content in the urine in psychoses and asserted that in psychoses, especially in conditions of depression, an increase of sugar is found in the urine. Maruyama investigated the blood sugar in psychoses in Japan and observed that in certain cases of psychosis there is an increased blood sugar content, but he was unable to find a particular relation between the psychoses and the blood sugar content.

The author reports his studies on the blood sugar tolerance in psychoses. The technic was as follows: 100 gm. of sugar were given to each patient in 300 gm. of water. Blood specimens were taken one-half hour before the sugar was given, and then from one to five hours after the ingestion of the sugar. The determinations were made by Bang's new micro method. An increase in blood sugar was found in conditions of excitement and a decrease in depressions.

The results obtained in a study of the blood sugar curves in five normal patients and in patients with psychoses may be summarized as follows:

1. Normal persons show the highest sugar content one hour after the ingestion of the sugar. After three hours, the blood sugar content is the same as before ingestion, and after five hours it has fallen to 0.09 per cent.

2. Epileptic patients show about the same course, except that the maximum at the end of one and five hours is higher than in the normal person.

3. Idiots show a rise in sugar content at the first hour after the ingestion of sugar, and they maintain this level for three hours. The level falls gradually to normal by the end of the fifth hour.

4. Schizophrenic persons show the highest blood sugar level at the end of the first hour, and then maintain this level for four hours.

5. The cases of dementia paralytica and manic-depressive psychosis show findings different from any of the others. The blood sugar content rises after one hour, just as in the other conditions. After two hours it falls, only to rise again to a higher level than ever before from the third hour.

On the basis that in dementia paralytica the cortex of the suprarenals is increased, and on the basis that mention is often made of the relation of schizophrenia and manic-depressive psychosis to the glands of internal secretion, the author feels that the findings tabulated above are in some way related to the internal secretions.

ALPERS, Philadelphia.

PATHOLOGIC ANATOMY OF THE COMBINED SUB-ACUTE DEGENERATION OF THE SPINAL CORD. I. BERTRAND and A. FERRARO, Il Cervello **3**:1 (Feb.) 1924.

This is a thorough histopathologic study of a case, which had previously been described clinically by Marie and P. Bailey under the title of "Combined Subacute Degeneration of the Spinal Cord." (Dégéneration combinée subaiguë de la moelle, Séance Soc. de Neurol., March 9, 1922). The patient, 65 years of age, showed intra vitam a paraplegia, first spastic and then flaccid, sensory changes and profound (not pernicious) anemia. Death from bronchopneumonia occurred about a year after the appearance of the first symptoms. Necropsy showed a small cancer of the posterior gastric wall, with metastasis in the liver and in the splenic and aortic ganglia, whose histologic appearance was that of an atypical epithelioma. No metastasis was found in the meninges, nor the different portions of the central nervous system. The spinal cord was found considerably reduced in volume, especially at the level of the dorsal segments. The histopathologic examination of the central nervous system carried out in all its details failed to show any sign suggesting an inflammatory process. It is briefly summarized by the authors in the following way:

This case presents a complex process of degeneration very diffuse, although predominating in the spinal cord. There undoubtedly exists a secondary funicular degeneration, localized mostly in the fasciculi of Goll, in the cerebellar tracts and in a lesser degree in the crossed and direct pyramidal tract. There are also disseminated zones of degeneration in the spinal cord and in the brain cortex having no special topography nor definite limits. From the combination of these two processes, the one secondary, the other primary, presumably toxic, an anatomopathologic complex results, which only imperfectly may be defined "combined degeneration." The perivascular changes are solely related to nervous desintegration, in conformity with the so-called law of the "neuro-interstitial" duality. The true vessels are to be considered normal. The desintegrative process is preeminently ectodermic in type.

The authors conclude from the absence of vascular lesions in their case that the eventual alterations of the vascular apparatus in combined sclerosis must be considered secondary. They accept the toxic theory and attribute to the gastric epithelioma the blood dyscrasia which gave origin to the profound anemia and to the consecutive spinal and encephalic changes. In order to explain the particular pathologic localization in some well defined tracts, they accept the Aufbrauchstheorie of Edinger, recalled by Brouwer and Blauwkip. With these authors, they also agree in rejecting any term which may suggest the existence of an inflammatory process, such as funicular myelitis of Henneberg, and acute disseminated myelitis, first used by Nonne.

NACCARATI, New York.

THE APPLICATION OF EXPERIMENTAL FORM INTERPRETATION TO PSYCHANALYSIS. H. RORSCHACH, edited after the death of the author by EMIL OBERHOLZER, Ztschr. f. d. ges. Neurol. u. Psychiat. **82**:240-274, 1923.

The author gives a correct analysis of a patient he had never seen, by means of the interpretation of the responses and comments this patient made about certain pictures and designs which were shown to him. These answers were obtained by Oberholzer and sent to the writer. The patient's psychanalysis by Oberholzer corresponded closely to the analysis made by the author after he had interpreted and classified the responses to the designs. (It is to be regreted that the designs, which are evidently of an impressionistic nature, black and white mixed with colors, were not reproduced. Without them the publication, though comprehensible, is not as clear as it might otherwise be.)

NEYMANN, Chicago.

THE SOURCE OF HYSTERIA. KURK SCHNIDER, Ztschr. f. d. ges. Neurol. u. Psychiat. **82**:275-278, 1923.

Hysteria is described as a disease having its foundation in the psychic desire "to be more than one really is." For this reason the patients play-act. Three kinds of hysteria are described according to their motive, origin and mechanisms; reflex hysteria, expression hysteria and organ hysteria.

NEYMANN, Chicago.

LICHENOID SKIN ERUPTION, A PSYCHOGENIC DERMATOSIS. AUGUST HOMBURGER, Ztschr. f. d. ges. Neurol. u. Psychiat. **82**:105-116, 1923.

An exceedingly interesting psychanalysis is made of a woman who developed a dermatosis on the basis of a juvenile sexual trauma and a resultant subconscious conflict which was reenforced and exaggerated by two other conflicts of later life. The psychanalytic explanation was given by the patient herself without any of the usual guidance or attempts by the physician to direct the mental association processes. The complete recovery of the patient's condition followed the psychanalysis.

NEYMANN, Chicago.

PSYCHIC SYMPTOMS FOLLOWING ENCEPHALITIS. J. B. JOERGER, JR., Ztschr. f. d. ges. Neurol. u. Psychiat. **82**:117-121, 1923.

The author describes the ethical deterioration of several patients who developed a parkinsonian syndrome following epidemic encephalitis. The intellect of these patients remained quite intact.

NEYMANN, Chicago.

THE THERAPY OF ARTERIAL SPASMS. ERICK MEYER, Arch. f. Neurol. u. Psychiat., Festschrift für Constantin v. Monakow, **13**:440-446, 1923.

Abnormal arterial spasms in the central nervous system and heart may occur without an increase in the general blood pressure. The frequent and transitory sensory and motor disturbances suggesting cerebral attacks cannot be explained satisfactorily on persistent local anatomic lesions. Visual disturbances and Meniére's syndrome may be dependent on fluctuations in caliber of arterial vessels. The fear of using active vasodepressor drugs, such as nitrites, is not altogether unfounded, and the slowly working purin derivatives are often preferred.

For more than five years Meyer has used a method for the treatment of vascular hypertonia on an arteriosclerotic basis which he thinks is of some value. His investigation was prompted by the assertion of Buedingen that it was possible to alleviate anginal cardiac attacks by the infusion of hypertonic glucose solution. He gave from 200 to 500 c.c. of a 10 to 25 per cent. solution intravenously, the purpose being to nourish the heart muscle. It has since been learned that undernutrition as the cause of anginal attacks could not be proved. Furthermore, the method was not altogether without danger, and so did not gain general acceptance. Meyer, therefore, reduced the amount to 10 or 20 c.c. of a 20 per cent. solution of glucose given intravenously, following which there was excellent improvement. It was found that after the injection, the blood pressure fell regularly, subjective disturbances were diminished, and no harmful results occurred. The fall of pressure took place from thirty minutes to three hours after the injection, and sometimes lasted several days. Anginal attacks and intermittent claudication disappeared for variable periods of time.

Normal serum or plasma produces vasoconstriction when it is used for the transfussion of calves' or rabbits' hearts. After the addition of grape sugar, the smaller arteries and capillaries of the coronary circulation become dilated. The addition of Ringer's solution or blood plasma to the sugar seems essential. The dilatation does not occur following the addition of cane sugar. Levulose, however, produced a response like that of glucose. In cases of contracted kidney with high blood pressure, no fall in the pressure occurred.

WOLTMAN, Rochester, Minn.

A TYPE OF PROGRESSIVE MUSCULAR ATROPHY WITH SUBACUTE COURSE. A. SOUQUES and T. ALAJOUANINE, Ann. de méd. **15**:281 (April) 1924.

The authors report a case of progressive muscular atrophy in a young man, aged 17, which began in the right hand and arm, extended to the left hand and arm, then to the lower extremities and finally to the facial muscles. The case was typical of progressive muscular atrophy but ran an unusually rapid course with only eight months between the onset of symptoms and the fatal termination, which was marked by acute bulbar symptoms.

Emulsions of the cervical cord and the medulla were introduced intracerebrally into four guinea-pigs and two rabbits. One of the rabbits developed symptoms but not until six months after inoculation. The right hind leg was affected first, then the opposite leg and finally the fore legs. There was no true paralysis but a muscular atrophy and trophic changes in the affected limbs. There was marked loss of weight. The animal was killed, and emulsions of the cervical and lumbar cords were injected into eight other rabbits. Seven of these died of intercurrent infections. The eighth animal showed no symptoms in the course of eight months.

A study of the lesions in the central nervous system of the patient showed them to be located chiefly in the gray matter of the cord, especially affecting the motor cells of the anterior horns and of the lower portion of the medulla oblongata. The anterior horn cells were diminished in number; in places, atrophic cells were scattered among normal cells. In the upper part of the cervical cord and in the lower part of the medulla, there was perivascular infiltration, especially marked at the decussation of the pyramidal tracts. The lesions in the rabbits were similar.

These pathologic findings and animal experiments indicate that the progressive muscular atrophy in this case was due to a neurotropic virus with a long

incubation period. They were unable to draw any conclusion regarding the virus, whether it is filtrable or whether it is a new specific form or a variant of some other known virus. They believe that the long incubation period distinguishes it from the viruses of rabies, of epidemic encephalitis and of poliomyelitis.

DAVIS, New York.

PSYCHOPATHOLOGY AND GENERAL PATHOLOGY. A. MAEDER, L'Encephale **19**:163 (March) 1924.

The author pleads the necessity for a general scheme of pathology embracing the anatomic, physiologic and psychic factors of man in their proper relationship. The advancement of knowledge in the various special branches of medicine has reached a point where it is wise to correlate and integrate the information acquired by these various departments of science. There seems to be a tendency to forget that each of the special branches of medical science is dealing with a rather complex animal, and that a proper understanding of this animal-man is dependent on a broad point of view.

The major portion of the paper is concerned with the inter-relation of the psychic and physical factors in disease. The author emphasizes that in certain types of persons, psychic conflicts produce vegetative neuroses and "functional" physical disease which at times may lead to "organic" disease. He points out that in chronic organic physical disease therapeutic measures may, in some patients, be futile because they find their physical disability to be advantageous. Proper psychotherapy is especially indicated in such cases if physicians wish to save their patients from charlatans.

The author maintains that in considering the relation between mind and body, attention must be especially drawn to the phenomena under investigation in psychic research. He states that after discarding phenomena accounted for either by fraud or by activity of the subconscious, there is a body of facts which rather suggests the necessity for departure from the present order of biologic and psychologic concepts. As for the "cures" occurring among the adherents of various occult and religious cults, Maeder is inclined to believe that the usual scientific dictum that the results are due to suggestion is inadequate as an explanation. He is led to wonder whether there is not some unknown spiritual factor—using the word spiritual in its broad sense—which is high in the hierarchy of psychophysical relationships.

The author closes his paper with an enumeration of certain interesting analogies between phenomena of physical pathology and psychopathology.

HYSLOP, New York.

PSYCHOPATHOLOGY AND GENERAL PATHOLOGY. A. MAEDER, Ztschr. f. d. ges. Neurol. u. Psychiat. **82**:176-192, 1923.

The contribution makes a plea for a correlation between the psychologic and physiologic factors in disease. The disturbances of the functions of the sympathetic nervous system and the resultant endocrine disfunctions caused by psychic conflicts are discussed. In melancholia, for example, it is stated that the conscious personality has fallen into the domination of a destructive internal psychic power. This hinders and disrupts the physiologic functions of the intestines, circulatory system, metabolism, etc. There is a certain analogy between inflammation, fever and immunity on the one hand and repentance, insight and atonement on the other hand. The former tends toward a physical

restoration of the body, the latter toward a renewal of the integration of the personality. The well-known self cure of Benvenuto Cellini is cited as an example.

NEYMANN, Chicago.

PROBLEMS OF CHARACTER IN THE LIGHT OF PSYCHIATRIC AND GENEALOGIC INVESTIGATIONS OF HEREDITY (WITH A SPECIAL EMPHASIS ON EPILEPTOID STATES). FRANCESKA MINKOWSKA, Ztschr. f. d. ges. Neurol. u. Psychiat. **82**:199-211, 1923.

This is a genealogic study of the ancestry of two patients of Bleuler's clinic. The two patients were suffering from a typical recurring schizophrenia with manic-depressive features. The paternal ancestors were found to be schizoid, the maternal ancestors were epileptoid in strain. These genealogic studies embrace seven generations.

NEYMANN, Chicago.

BELLADONNA DELIRIUM. SOPHIE MORGENSTERN, Ztschr. f. d. ges. Neurol. u. Psychiat. **82**:231-239, 1923.

This is a description of the mental and physical symptoms of two patients who had eaten atropin berries with suicidal intent. Both recovered after several weeks of delirium.

NEYMANN, Chicago.

A STUDY OF THE PHYSIOLOGICAL EFFECTS OF CAFFEIN UPON PLANARIA DOROTOCEPHALA. MARIE A. HINRICHS, J. Exper. Zool. **40**:271 (Aug. 5) 1924.

The physiologic action of caffein on the fresh-water triclad, *Planaria dorotocephala,* was investigated by four methods. The nervous system in this form is not highly specialized. The data on oxygen consumption by the Winkler method, the comparative estimation of rates of carbon dioxid production, and the head-frequency method of Child showed that caffein may act as a stimulating or as a depressing agent, the effect depending on the concentration employed and on the length of period of exposure. In all experiments, the initial effect was a marked stimulation, which changed to depression when the exposure was continued long enough. Regional and individual differential susceptibility to caffein was in general similar to that observed with other agents. With decreasing concentrations, the nervous factor in the action of caffein probably played a large part. The stimulation was more extreme, and death occurred relatively earlier in the anterior zooid, in which nervous structure and function are fully developed, than in the posterior zooid, in which regressive changes occur. As regards the difference between margins and median region, it appeared to be true that the sensory, motor and glandular specialization of the margins is relatively high, and that the more extreme the stimulating action of an agent, the greater is the differential between margins and median regions. Caffein apparently acts to some extent as a general protoplasmic stimulant or depressant in *Planaria dorotocephala,* but some degree of selective action on the nervous system evidently occurs.

WYMAN, Boston.

CASUISTRY OF NERVOUS TIC. E. SIGG, Ztschr. f. d. ges. Neurol. u. Psychiat. **82**:279-285, 1923.

This is a description of the psychanalysis and cure of three cases of nervous tic.

NEYMANN, Chicago.

MORAL OLIGOPHRENIA AND SCHIZOIDIA. JOHN E. STACHELIN, Ztschr. f. d. ges. Neurol. u. Psychiat. **82**:286-291, 1923.

This contribution points out that moral imbecility or moral oligophrenia would seem to be closely allied to the schizoid type. Syntonic personalities never seem to show a grave lack of moral sense, although their carelessness and carefreeness may at times get them into legal difficulties.

NEYMANN, Chicago.

NEUROLOGIC EXAMINATIONS OF SCHIZOPHRENICS. HANS STECK, Ztschr. f. d. ges. Neurol. u. Psychiat. **82**:292-329, 1923.

Based on the examination of 400 cases of dementia praecox, an attempt is made to associate this disease with some of the symptoms found in postencephalitic states. The fact that catatonia in particular often shows analogous symptoms to those found in the parkinsonian syndrome is dwelt on. Such neurologic symptoms are the Lévi-Mayer joint reflexes, the snout reflex, the salve face, the reduction of skin reflexes, the exaggeration of deep reflexes and the general idiomuscular excitability. Patients with paranoid dementia praecox occasionally show these symptoms, hebephrenic patients show them more often, and they abound in catatonic states. The conclusion is therefore reached that there must be some involvement of the basal ganglia in many cases of dementia praecox, especially in catatonia. A finer anatomic localization is not attempted. The prime cause in such an involvement, be it psychic or physical, is left to the reader's judgment.

NEYMANN, Chicago.

ROENTGEN-RAY THERAPY OF POLIOMYELITIS: PRELIMINARY REPORT. HERMAN B. PHILIPS and WALTER I. GALLAND, J. A. M. A. **82**:1847 (June 7) 1924.

Hyperesthesia as a dominant symptom is characteristic of the polyneuritic type of poliomyelitis. The authors noted the marked persistence of this symptom weeks and even months after the subsidence of the acute stadium. The pain of which these patients complained added considerably to the difficulties of early treatment.

In a previous article, one of the authors called attention to the therapeutic advantages of stimulating doses of roentgen ray. Special attention was called to the analgesic and antispasmodic effects of this type of therapy, as well as to the curative effects. The gratifying and frequently spectacular results led them to apply stimulating doses of roentgen irradiation in recalcitrant cases of poliomyelitis.

The types of cases treated may be divided into two general classes—one in which stimulation of the diseased anterior horn cells was desired, with the objective of improvement of the paralysis, and a second, in which the irradiation was given to overcome the persistent hyperesthesias so far unresponsive to any form of therapy. In the latter group, four patients exhibiting marked persistent hyperesthesia were irradiated with extremely encouraging results. All of these patients showed marked improvement within a week after the first irradiation of the involved spinal segments.

NIXON, San Francisco.

SENSORY PARAMUSIA. HENRY MARCUS, Ztschr. f. d. ges. Neurol. u. Psychiat. **81**:625-644, 1923.

The author describes a case of sensory acustic paramusia and partial tone deafness. The patient was an 80 year old successful music teacher. Several

years previous to her final sickness she began to have peculiar ideas, and stated that she believed in clairvoyance. For several months previous to her death she described marked auditory hallucinations. Three years previous to this time, the patient began to show lack of appreciation of music and claimed her scholars always sang in the wrong pitch. From time to time she attended concerts but was always dissatisfied with the productions, saying that the music was poor, and being especially discontented with vocal music.

The gross pathology of the brain in this case showed atrophy of the anterior two thirds of the first left temporal gyrus, while the posterior third did not show any pathologic findings. The microscopic examination of the atrophic portion showed decided atrophy of the cortex with proliferation of the glia, senile plaques and findings in many ways similar to influenza encephalitis. From this case as well as from two other cases which he cites from the literature, the author localizes the sensory music sense in the anterior two thirds of the first left temporal gyrus.

NEYMANN, Chicago.

OBSTETRIC BRACHIAL PARALYSIS (ERB'S PALSY). SAMUEL W. BOORSTEIN, J. A. M. A. **82**:862 (March 15) 1924.

The author states that if these cases are treated early and properly, one may expect in the mild cases a good recovery in three or four months. The more severe cases will require about six or seven months for a complete recovery.

The important treatments are: (1) early support, (2) support in the proper position to prevent deformities, (3) continuation of support for a considerable period, and (4) massage and exercises carefully carried out.

Electricity not only has no beneficial effect on the child, but it is harmful in that it prevents voluntary movements and causes overuse of the denervated muscles.

Nerve operations are indicated if no advance is made in four months. After that period, if sufficient improvement is noticed one may wait four months more, provided, of course, proper orthopedic treatments are continued.

NIXON, San Francisco.

Book Reviews

LE RADIO-DIAGNOSTIC DES AFFECTIONS INTRA-RACHIDIENNES PAR LE LIPIODOL SOUS-ARACHNOIDIEN. LA FORME PSEUDO-POTTIQUE DES TUMEURS INTRA-RACHIDIENNES. By LOUIS LAPLANE, M.D., Paris: Amédée Legrand, 1924. Pp. 281.

That the use of an iodized poppy oil may be exceedingly valuable in localizing spinal cord tumors and other lesions causing spinal block, is certain. Little concerning the procedure has appeared in the literature outside of France. This monograph, of 281 pages and 62 illustrations, by LaPlane, who has worked with Sicard for the last two years, fully presents the subject in its various aspects.

The iodized oil (1 or 2 c.c.) is introduced into the cisterna magna, or the subdural space at some lower point, and is allowed to gravitate downward. A roentgenogram shows where it is arrested.

Quite properly the author begins by considering the effect of the injection itself. (We may state that there has been some difference of opinion on this point.) In the vast majority of cases, the injection is painless. Exceptionally, there is pain, one or more of three kinds. First, if there is compression of the cord or spinal block, the iodized oil may cause pain apparently arising in the sensory roots at the point of the lesion. This pain is of short duration. Second, there may be pain coming on several hours after the injection, felt in the legs, especially in the sciatic area, and lasting for several days. It may be rather severe or only a sense of discomfort. This pain disappears when the patient lies down. Third, occasionally there is severe pain felt at the base of the coccyx. This is not relieved by the recumbent posture, and the coccyx is tender to palpation.

Frequently, there is some rise of temperature with headache after the injection. Ordinarily the fever disappears in three days; it rarely lasts four or five days. In two cases (out of more than 300), the fever reached 40 C. (104 F.) with signs of meningeal irritation, but all symptoms disappeared in three days. Generally, the injection causes mild lymphocytosis of the spinal fluid.

In case the iodized vegetable oil is not caught by an obstructing lesion, it at once descends to the dural cul-de-sac, where it remains indefinitely — at least for months and even years — causing no disturbance.

The author was able to examine postmortem five subjects who had received the iodized oil from one and a half to eleven months before. In four of the five, as usually happens, the iodized oil had become "fixed" in the cul-de-sac. During life, this immobility of the globule of iodized oil can be demonstrated by the roentgen ray. In three of the four, the iodized oil was held in place by an exceedingly thin envelop of fibrin, without signs of inflammation. In the fourth, the oil was enclosed by a cystlike wall.

Occasionally, the iodized oil does not descend quickly and smoothly, even when there is nothing abnormal in the spinal canal. The author thinks that this is due to defects in the preparation. The oil must be pure and of proper consistence and the proportion of iodin should be 45 per cent. A preparation that is or becomes brown, should be discarded.

Chapter III is an adequate anatomic survey of the subarachnoid space from the cisterna magna to its inferior termination. This study embraces a number of measurements relating to the cisterna magna and the suboccipital region, including the effect of flexion and extension of the head on topographic relations.

Chapter IV contains a full description of the technic of the injection at different levels, and in Chapter V is graphically described the descent of the iodized oil as seen with the fluoroscope. About three minutes are required for the passage from cisterna magna to sacrum, and the lateral decubitus on an incline is more favorable than a sitting position. The roentgenograms of the iodized oil in a normal person under different conditions and in different postures are then described.

Chapter VI gives instructions as to method and detailed results in various lesions. These are illustrated by cases, roentgenograms and diagrammatic drawings. Chapter VII covers indications, results and limits of the test.

The second part of this monograph (ninety-six pages) is on the simulation of spinal caries by spinal canal tumors (La forme pseudo-pottique des tumeurs intra-rachidien). It is in effect an effort to promote the diagnosis of intraspinal tumors in the preparalytic stage and is based on the study of nine cases; six of the cauda equina, two dorsal and one cervical. Symptomatology and differential diagnosis are thoroughly discussed, especially as to spinal fluid findings and the use of iodized oil. There is a chapter on indications for laminectomy, in which one sadly misses a consideration of the enormous experience and notable results of English and American surgeons. Some excellent case reports and conclusions close the volume. It is a valuable monograph which every neurologist and every neurologic surgeon should have — and use.

THE EVOLUTION OF MAN: ESSAYS. By G. ELLIOT SMITH, M.A., M.D., Litt.D., D.Sc., F.R.C.P., F.R.S. Humphrey Milford, Oxford University Press

Few large tomes contain as much neurologic food for thought as this little book of three chapters by Professor Elliot Smith. As the author says, it is impossible to understand how human intelligence came into being until one has some clear conception of the sequence of changes through which the ancestors of the human family passed in their progress toward this attainment of man's estate. And it is just this emergence of the qualities of mind that particularly interests the author. To him it is the process of building up the cerebral cortex that has allowed man to attain his extensive power of discrimination, skill and understanding. He has therefore employed the facts of the evolution of the brain to unite into one comprehensive story the various data concerning man's pedigree. It is the breadth of the author's learning that makes the book so interesting, bringing together the facts gathered from such diverse departments of knowledge as zoology, comparative anatomy, geology, paleontology, physiology and psychology.

The "foreword" is a sketch of man's pedigree. The principle anthropologic data are summarized and illustrated by two clear diagrams. In the first chapter, the evolution of man is discussed more in detail, the steady and uniform development of the brain along a well defined course, giving us the fundamental reason for "man's emergence and ascent." It is impossible to abstract for a review the argument of such a closely reasoned chapter, but one part is perhaps more striking and illuminating than the rest — the author's attitude toward differentiation and specialization. Most of us have looked on man as the most highly

specialized product of evolution, in whom there are retained certain primitive characters that are rather degrading. To Professor Elliot Smith, the retention of primitive characters is not an indication of lowliness; rather is high specialization a confession of weakness. The stock from which man emerged played a humble rôle for long ages, while other stocks became specialized, surpassed it and succumbed. But man's ancestors maintained the plasticity of a generalized structure, free to develop a well balanced use of limb, body and brain. Thus the shrews which took to an arboreal life underwent a brain change which diminished the size of their olfactory apparatus and greatly increased the size of the areas given up to visual, tactile, acoustic, kinesthetic and motor functions. The result was a more equable balance of the senses, and the development of a mechanism for mutually blending them in consciousness. From this humble beginning, the development of the great prefrontal area of the human brain can be traced through the primates to man, and with the development of right-handedness and gestures, we approach symbolization and speech, the last and greatest impetus to intellectual development.

The second chapter takes up primitive man, and discusses in some detail the known remains and the relationships of primitive man physically and psychologically to the extant savage races. To the medical reader, it is of great interest to obtain from a biologist an opinion on a psychologic problem like that of the effects of environment and training. In Elliot Smith's opinion, any human being, if trained from earliest youth, could attain the skill of the native tracker in observing with all his sense organs and interpreting the observations. No wonder the men of primitive races now living take little interest in our complex civilization, when they have spent all their years of training in the study of forest signs and forest life. Such lack of interest in the "arts of civilization" is not an indication of inferiority on the part of the savage — it is merely an accident of environment. The mind of man does not have to pass through "cultural epochs," it merely has to come easily into contact with accumulations of knowledge.

The third chapter, entitled "The Human Brain," is a short, well illustrated essay on the functional anatomy of the cerebral cortex from the standpoint of paleontology and comparative anatomy. Mammals alone have acquired a neopallium, an instrument with almost unlimited potentialities for the cultivation of skilled movements. These of course depend on a variety of sense organs for information, but the neopallium is the amazing machine for integration. Even more remarkable is the mechanism that can record the results of past experience, allowing behavior to be modified in the light of knowledge. This also allows a high degree of automatism to be acquired by training, which is perhaps the most essential factor in the attainment of high degrees of skill. Thus the author traces the development of the dominant sense of vision in man's ancestors, and shows how its use and improvement led to stereoscopic vision and conjugate eye movements. These were logically followed by increased powers of discrimination, and led eventually to esthetic sense.

It was then the attainment of the realization of space and time, and the faculty of recognizing objects by their shape, color, size and texture that marked the transformation of ape into man. These abilities made it useful for him to devise names for things, and thus arose language, vastly increasing the capacity for thinking in symbols.

We will not relegate this delightful book to dusty shelves by saying that it should be in every medical library, but we will say with emphasis that it succintly gives information and expresses a point of view with which every neurologist and psychiatrist should be acquainted.

WOMEN CHARACTERS IN RICHARD WAGNER. By LOUISE BRINK, PH.D. Pp. 125. Washington, D. C., Nervous and Mental Disease Publishing Co., 1924.

If this monograph had reached us with a conventional cover design picturing Wotan's farewell from Brunhilde, under the title "Sidelights from the Ring of the Niebelungs" by Richard Wagner, we might have enjoyed rereading some of the more significant lines of Wagner's questionable poetry, and found some interest in the citation of excerpts from various psychanalytic writings. To submit material found in this monograph under the guise of a contribution either to literature or to the study of mental disease is, to put it mildly, unjustifiable. If an analysis of the unconscious formulation of character in folklore was to be attempted, it might have been well for the writer to resort to the original series of the Saga, the Edda, or at least to the medieval tradition of the Ring. To interpose a conception of Wagner's, has nullified this aspect. No writer has been more deliberate and conscious than Wagner, and the trilogy is a deliberately constructed device to support a definite musical composition, anything in the prose writings of Wagner notwithstanding. That his conscious and deliberate misrepresentations have penetrated even into his own autobiography posthumously published, has been generally acknowledged, and requires no further proof. It appears that the well-known vagaries of Wagner's style have affected the author, even though the statement is made that she was impressed with his clearness of expression.

This little treatise consists principally of a loosely constructed jumble of citations from Wagner's prose works and lengthy reprints from the Ring of the Niebelungs, interspersed with rather irrelevant quotations from psychanalytic work. To retell the story of various attachments of a growing child by the use of a few terms used in psychanalytic literature is not to make a contribution to psychanalytic thought. In its entirety, it is not an attractive retelling of the story of the Ring, with a forced and unsuccessful attempt to bring it into relation with some fundamental conceptions of psychanalysis. The writer's naive exposition of the psychanalytic idea at the very outset is characteristic of the lack of seriousness of the entire contribution. In her foreword she asks: "Does it seem fanciful to read such an elemental life story into these elaborate Dramas?" It does in fact not seem fanciful, but is rather proof of the complete lack of imagination and interpretative thought. It would be useless to cite at length the platitudes that seem to be the only contribution of the writer. What can we say about such statements as "Psychanalytic research recognizes that the artist's cravings are simply those of all men?" Is it a discovery of psychanalysis that artists are human? The statement that investigations are being carried on in the most hidden recesses of the unconscious mental life is the sort of generalization that is most detrimental to the consideration by serious scientists of the clear exposition of the psychanalytic writings of Freud, who is so eminently frank in acknowledging the limitations of his discovery. The most bombastic generalizations with which the prose works of Wagner abound have been chosen. By far the major portion of this work consists, as we have already stated, of reprints of one kind or another, and the originals have been reviewed in their proper places.

The one absolute postulate of the Freudian idea is iron sincerity. Dilettantism is a grave danger, and those of us who believe in the development of psychanalysis for practical application in neuropsychiatry cannot be too emphatic in objecting to anything that is not dictated by clarity of thought and sincerity of intention. The author's apology for not going into the psych-

analysis of the music of the Ring is gratefully accepted. If clarity and definiteness of thought are not obtained in the interpretation of the written work, we are pleased to be spared similar unravelings of the musical concepts.

INTERNATIONAL CLINICS. VOLUME III. THIRTY-FOURTH SERIES, 1924. Pp. 304. Philadelphia: J. B. Lippincott Company, 1924.

This volume maintains the high standard long ago established for these publications. The contents are divided into six sections: "Public Health and Hygiene," six papers; "Diagnosis and Treatment," seven lectures or papers; "Medicine," four papers; "Pediatrics," one paper; "Surgery," one paper; "Obstetrics," one paper.

Of direct interest to neurologists are a lecture on tic by Morris Grossman, a paper on the parkinsonian syndrome by Alfred Gordon and a paper by Van Leeuwen of Leiden on recent advances in the treatment of bronchial asthma, hay-fever, urticaria, eczema, Quincke's edema, migraine and epilepsy. Concerning the last two, the author says: "In any case the fact that allergy may sometimes result in attacks of epilepsy and migraine is not to be denied," but he does not claim brilliant results from anti-allergic treatment of these disorders.

Many of the other papers are interesting and instructive. This volume, like its predecessors, is of value to the general practitioner and to such others as wish to keep informed in fields other than that of their limited work.

LEITFADEN FÜR DIE UNTERSUCHUNGEN UND DIAGNOSTIK DER WICHTIGSTEN NERVENKRANKHEITEN. By PROF. DR. ADOLF STRÜMPELL. Leipzig: F. C. W. Vogel, 1924.

It is surprising that such a distinguished neurologist as Professor Strümpell should write such a small book for beginners in neurology. On the other hand, no one could have done it better. It is a brief discussion, containing only 151 pages.

The book is divided into three parts. The first takes up the "Method of Examination of Nervous Diseases;" the second, the "Diagnosis;" the third, "Special Diagnosis of Some of the Organic Nervous Diseases." Functional conditions are not discussed. In the discussion of the clinical syndromes, the pathology is touched on lightly. Necessary clinical discussion is brief.

It is interesting to observe that under the heading of the different forms of progressive atrophy there are five classifications: (1) different forms of progressive muscle atrophies, (2) hereditary ataxia (Friedreich's ataxia), (3) hereditary spastic spinal paralysis, (4) chronic familial chorea (Huntington's chorea) and (5) Wilson's disease.

NOTICE

SUBSCRIPTIONS NOW DUE FOR 1925

Do not overlook the blue slip inserted in this issue of Archives of Neurology and Psychiatry. It is a bill for your subscription for the coming year. If you have paid in advance, disregard the notice.

Printed on the slip you will find the names and subscription prices of the other journals published by the American Medical Association; also blank lines wherein may be written the names of the other journals for which you subscribe. The slip affords a convenient means of caring for several remittances by one check or draft.

Because of increased publication costs and also because of contemplated improvements in this and other special journals of the Association, the Board of Trustees has found it necessary to increase the subscription price of some of these publications. The annual subscription price of Archives of Neurology and Psychiatry has been fixed at $8.00, effective January 1, 1925.

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